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OPERATIVE RESULTS IN TWO HUNDRED AND ELEVEN CASES OF CONVERGENT STRABISMUS

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AND
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NEW YORK

It is a matter of common knowledge that the operative results in strabismus are extremely variable. Some surgeons are highly pleased with their results, while others of equal skill are disappointed in the outcome. Such a difference in opinion does not necessarily mean a wide variation in the results but rather means a discrepancy in their evaluation. The surgeon who routinely expects binocular single vision with complete abolition of the deviation is faced with many disappointments, while the operator who is satisfied with an immediate cosmetic correction without thought of preservation of convergence or of adequate rotation is lulled into a false sense of security about his operations on muscles. Between these two extremes there is a middle ground into which most surgical corrections fall; the results are not perfect, since binocular single vision is not restored, but the correction does not cripple the movements of the eye and is most satisfying to both the patient and the surgeon.

The inability to predict a certain effect from a given procedure is dependent on many variable factors. Differences in the size, strength and insertion of the muscles are overemphasized by many surgeons but must be considered. The technical difficulties attendant on the determination of the exact amount of shortening or of lengthening done in a given case are another reason for the failure of surgeons to reduce muscle surgery to a mathematical formula. Without entering into a lengthy discussion on the merits of these and other variants, we find it sufficient to say that they do exist and that the mathematical millenium is not in

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Ext.

sight. The experiences herein recorded leave much to be desired, but it is hoped that they will stimulate other surgeons to turn the searchlight on their results.

During the period covered by this analysis, 349 patients were operated on for a muscular anomaly. The deviation was inward in 239, outward in 85 and only vertical in 25. With many of the lateral deviations there was also a small vertical one, but these figures show the relative frequency of the type of deviations for which the patients sought help. In other words, we operated twice as often for convergent strabismus as for all the other types combined. Because of this increased frequency we shall confine our discussion to the cases of convergent strabismus. Not included in this study are 25 in which one or more operations had been done elsewhere and 3 in which the data were incomplete.

Analysis of the remaining 211 cases of convergent strabismus revealed certain interesting facts. There was almost the same number of male as of female patients, 107 and 104, respectively. In 128 cases the strabismus was definitely monocular. The right eye was the squinting eye in 59 cases and the left in 69. Alternating fixation occurred in 83.

ANALYSIS OF CASES

Age of Onset.—The exact age of onset is not always easy to elicit, but in 28 per cent of the cases the strabismus was present from birth. It appeared prior to the age of 4 in 80 per cent, and in only 10 per cent did it occur after the sixth year. The average age at onset was 1.9 years. The age at which the squint appears bears no relationship to the severity of the deviation. All degrees of strabismus were encountered in all age groups, although most of the strabismus occurring early in life or present at birth was severe. Slight deviations could easily be overlooked in young children, and probably only the children with severe deviation were brought for treatment.

Vision.—A difference in visual acuity of two lines or more was present in 108 cases. Such a discrepancy was arbitrarily selected as representing amblyopia ex anopsia. The original vision of the squinting eye was less than 20/100 in 54 cases, which indicates that one can expect a similar visual acuity in about 1 out of 4 cases of untreated convergent strabismus. For various reasons, in 60 of the 108 cases in which there was amblyopia, occlusion was not considered advisable. In 41 of these 60 the age of the patient (over 7) plus the severity of the amblyopia at the time of the first examination made it impractical to carry out prolonged constant occlusion, while in 8 others, in which there was nystagmus, treatment was considered unwise. In the others (11) the patient had previously used occlusion and refused to submit to further

trial. Of the remaining 48 cases it was conscientiously employed in only 31. Nothing short of constant complete occlusion was found to be effective, and this was not always successful, for vision of 20/50 or better was obtained in only 23 (70.9 per cent) of the 31 cases in which this treatment was given a fair trial. The number of cases in which occlusion was employed may be misleading, because this analysis covers cases observed as long as eighteen years ago, before occlusion was adopted as a routine procedure.

Refractive Error.—This was determined during complete cycloplegia by the use of the retinoscope. Analysis of the refractive error was figured by adding one-half the astigmatism to the sphere, and the larger error of the two eyes was recorded. In only 17 per cent of the cases was the refractive error over 4 D. of hyperopia, and in 68 per cent it was under 3 D. Marked anisometropia was rarely encountered, being 2 D. or more in only 7 cases. The relation of the refractive error to the amblyopia was studied without a definite conclusion being reached. In some of the cases of the most severe amblyopia it was found associated with minimal refractive errors. The average amount of hyperopia present in the cases of amblyopia was 4.2 D., as compared with 3 D. for the entire group.

No relationship was found to exist between the amount of deviation and the refractive error. For example, in 5 cases in which there was less than 1 D. of hyperopia there was 50 prism diopters of esotropia, and in 5 in which there was 6 D. of hyperopia there was squint of less than 50 prism diopters. Small and large deviations were encountered with all types of refractive errors. The degree of the refractive error, therefore, seems to have little bearing on the amount of the deviation.

Deviation.—The deviation was measured whenever possible with the screen test for both distance and near vision, and if the results were different the greater was taken. It is interesting to note that in 71 per cent of the cases there was a deviation of 40 prism diopters or more and the limits of the deviation varied from 15 to 90 prism diopters. Almost the same average deviation, 47 prism diopters, was found in all age groups and with all refractive errors.

OPERATIVE RESULTS

In spite of the inadequacy of an analysis of results based on measurements of the deviation before and after operation, we have selected it as the best available. An evaluation for statistical purposes of the results obtained is almost impossible. As is generally appreciated, the exact measurement of the final (postoperative) deviation in prism diopters falls far short of telling the state of mind of the patient or of his parents. Any one who has operated to correct strabismus has

learned that in many cases in which the surgeon finds undercorrection or overcorrection to a disquieting degree the patient is perfectly happy over the result. Conversely, in some cases in which there is only a few diopters of residual squint the patient is still dissatisfied. With the belief that we are still a long way from the goal of achieving binocular single vision and fusion in a high percentage of our cases, we are giving the results only in terms of the deviation. For this analysis we have arbitrarily selected a "cure" as a residual deviation of no more than 10 prism diopters for either distance or near vision. In 43 per cent of the cases one operation was sufficient to effect a "cure," while in 44 per cent a residual deviation of more than 10 prism diopters remained. The undercorrections varied in severity, but in most of them the residual deviation was approximately 20 prism diopters. It is interesting to note that a second operation was advised in only 18 instances of undercorrection and performed in 15. This would indicate that even though the primary result was not ideal in many instances it was not bad enough to warrant further surgical intervention. In 13 per cent of the cases some degree of overcorrection was obtained, but a corrective secondary operation was performed in only 3 cases. Not included in these groups are 23 per cent of the total series, because the postoperative follow-up was of insufficient length. In most of these cases the patient came from a distant city for operation and was referred back to his local oculist on discharge from the hospital.

There were 44 (20.8 per cent) of the 211 cases in which the result of the original operation was not satisfactory. A second operation was done in 18 of these, with a successful result in 13 (72.2 per cent). In 1 case it was necessary to operate three times. This leaves 31 cases (14.7 per cent) in which the result was classified as a failure; i. e., either the original deviation was not reduced by 50 per cent or an overcorrection of 10 or more prism diopters resulted.

A study of the records of the 26 "failures" in which no secondary operation was either recommended or performed does not show why this occurred, nor does it determine the cause of failure. The age of onset, the refractive error, the frequency of amblyopia, the degree of deviation and the age at operation all averaged about the same as they did for the entire series. While our data on the frequency of abnormal retinal correspondence are too incomplete on the series as a whole to be included in this study, this defect was no more frequent in the cases in which operation failed than in those in which it did not.

The average age at operation was 9.1 years, but in 64 per cent of the cases the operation was performed prior to this age. In 23 cases (14 per cent) it was done before the age of 3 years. Two patients were 13 months, 1 was 15 months and 1 was 18 months old. A little over half

(52 per cent) of all the patients were operated on between the ages of 3 and 8, while 34 per cent were older than 8. The oldest patient was a woman aged 44. In 79 per cent of our cases operation was performed within five years after the onset of the deviation. With the hope of finding the ideal age for operation, we divided the patients into three age groups and classified the operative results as shown in the accompanying table.

The 163 cases included in this analysis were only those in which the follow-up was adequate. From this study it appears that the operative results are comparable in all age groups with one exception, i. e. the high percentage (20 per cent) of overcorrections obtained in the intermediate age group. We have no explanation for this finding, but the increased frequency is unmistakable and worthy of further study.

In an effort to determine the amount of correction that can be expected from a given procedure the largest group was selected, i. e.

Results in Various Age Groups

Age	Undercorrection (Over 10 △)		Eyes Straight (Less than 10 △)		Overcorrection (Over 10 △)		Total	
	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent
3 years or under.....	10	43	12	52	1	5	23	14
3 to 8 years.....	34	40	34	40	17	20	85	52
Over 8 years.....	28	51	24	44	3	5	55	34
Total.....	72	44	70	43	21	13	163	

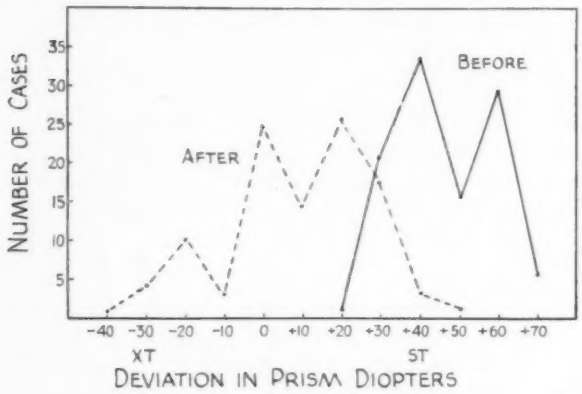
the cases (108) in which simultaneous resection of the external rectus muscle and recession of the internal rectus muscle of the same eye were performed. To eliminate as many variables as possible, only the cases in which the age at onset was definitely under 6 years, the follow-up was adequate and there were no complications (nystagmus) were included. The deviations before and after operation are shown in the two curves in the accompanying chart. The two curves are strikingly similar and show an average postoperative reduction in the deviation of almost 40 prism diopters.

No method has been devised for accurately measuring the exact amount of resection or recession done. The great majority of the operations were estimated to be 10 mm. resections and 4 mm. recessions, but these figures acquire meaning only when one is intimately acquainted with the method employed. It is therefore impossible to establish precise rules in muscle surgery. That a resection and a recession done on one eye and at the same operation will give an average reduction of 40 prism diopters is about as far as one can safely go toward a dogmatic statement. Such a statement has obvious disadvantages, yet it establishes a

base line which can serve as a valuable guide in determining what and how much to do to correct a given deviation.

Of the 108 cases in which a simultaneous resection and recession on the same eye were done the strabismus was classified as alternating in 53 and monocular in 55. The age at operation, the refractive error and the amount of deviation averaged practically the same in the two groups. The operative results are strikingly shown in the following tabulation:

	Alternating Strabismus, Percentage	Monocular Strabismus, Percentage
Undercorrection	66	23.5
Eyes straight	24.5	54.6
Overcorrection	9.5	21.9



Deviation before and after operation in 108 cases of combined resection and recession.

A study of these figures reveals that the results in the cases of alternating strabismus were definitely inferior to those obtained in the cases of monocular strabismus. An undercorrection was the rule in the former group, which would seem to indicate that alternating strabismus requires more extensive operative procedures. The increased frequency of an overcorrection among the cases of monocular strabismus points toward the need of greater conservatism in the treatment of this type of strabismus.

While a single recession was employed in only 11 cases, it was found that the average correction resulting from this procedure was 7.3 prism diopters. In a few cases in which there was a marked spasm of convergence a correction of 20 to 30 prism diopters was obtained. This

result not only serves to show the difficulty in predicting the effect from a given procedure but indicates that a single recession of 4 mm. or less is apt to be disappointing. A recession of more than 4 mm. was not used because of its crippling effect on the power of convergence. Bilateral recessions were employed too infrequently to permit any conclusions as to their efficacy.

TECHNIC

The technic employed in the resection was essentially that described by Reese many years ago. During the past eight years the suture material has been ten day 0000 chromic catgut, which was used in both the resection and the recession operation and for the conjunctival closure. In the resection we used two double-armed sutures passed first through the muscle from within out, then through the stump and the conjunctiva and finally through the other conjunctival edge. By this technic an inversion of the conjunctiva beneath the muscle stump is prevented, which always has been an objection, largely theoretic, to the Reese method of closure. In the recession two single-armed sutures were used according to a technic previously described. In all cases particular attention was paid to accurate closure of the conjunctival wound. The postoperative treatment consisted in bandaging only the eye operated on. The dressing was changed every forty-eight hours for approximately one week, after which the eye was left uncovered. No mydriatic was used except in rare instances. The patient was discharged from the hospital at the end of forty-eight to seventy-two hours. Most of the operations were performed with the patient under general anesthesia; general anesthesia was used particularly when a generous resection was desired.

POSTOPERATIVE COMPLICATIONS

A review of our cases reveals that no postoperative infection occurred; nor were there any alarming reactions. The complications encountered were 3 granulomas, the result of imperfect conjunctival closure, and 8 subconjunctival cysts. Six of the cysts developed at the site of the recession and were possibly produced by buried suture material. Some thickening was frequently seen over both the external and the internal rectus muscle incision, but in no instance was this sufficiently disfiguring to warrant concern.

Postoperative diplopia was frequent as a transient complaint, but only in 3 instances did it show any tendency to be permanent. In these 3 cases the patient was adult and had monocular squint. The vision of the affected eye was 20/70 in 1 and 20/200 in the other 2. In 2 of these cases the persistent diplopia was only slightly annoying, while in 1 case it was most distressing. Some residual convergent squint remained in 2, while an overcorrection was present in 1. The troublesome diplopia occurred in a woman of 44 who had vision of 20/200 in the squinting eye as a result of old corneal opacities. After three and one-half months the overcorrection was remedied by a secondary operation, with complete disappearance of the double vision.

SUMMARY

Analysis of 349 cases in which an operation was performed for a muscular anomaly showed that twice as many patients sought relief for an inward deviation as for all the other types of deviation combined. The average age at onset of convergent strabismus was found to be 1.9 years. Neither the age of onset nor the refractive error bore any relationship to the severity of the squint. Vision of 20/100 or less was present in 1 of 4 cases in which there had been no treatment. In 68 per cent of the cases of convergent squint the refractive error was less than 3 D. of hyperopia, and severe anisometropia was found in only 7 cases. The average amount of deviation in all age groups was 47 prism diopters. A simultaneous resection and recession on the same eye produced on the average 40 prism diopters of correction. The results with this combined operation were definitely inferior in those cases in which there was alternate fixation. In 43 per cent of 163 cases one operation was sufficient to reduce the deviation to 10 prism diopters or less, while an undercorrection was obtained in 44 per cent and an overcorrection in 13 per cent. Of those cases in which a secondary operation was performed a satisfactory result was obtained in 72.2 per cent.

The age at the time of operation seemed to have no bearing on the result except that there was an increased tendency toward production of an overcorrection in patients operated on between the ages of 3 and 8 years.

Postoperative complications were rare and consisted of 8 subconjunctival cysts and 3 granulomas of the conjunctiva. In 3 cases there was persistent postoperative diplopia, but it was distressing in only 1.

30 West Fifty-Ninth Street.

DISCUSSION

DR. HENDRIE W. GRANT, St. Paul: This excellent analysis of 211 cases of convergent strabismus corrected surgically should receive the careful consideration of every one concerned with the problem. It is of real value because it shows the possibility of producing satisfactory results in spite of amblyopia, poor fusion, overcorrection and undercorrection and inability of the patient to obtain binocular single vision.

The inability to restore binocular single vision is worthy of further emphasis. In its evolution convergent strabismus arises from divergence insufficiency or convergence excess. The divergence insufficiency type is infrequent and arises from a spasm of the internal rectus muscle when the eyes are focused on a distant object. The characteristics of this type are intermittent strabismus of low degree without amblyopia, normal retinal correspondence, a fair degree of fusion and preservation of the convergence function. Surgical correction, when necessary, usually results in spontaneous binocular fixation and some degree of binocular single vision. I have not observed false retinal correspondence in the patients examined.

Commonly, convergent strabismus occurs as a result of convergence excess and is of sudden onset, and structural changes in the muscles are often pronounced. The characteristics of this type are amblyopia (in over 50 per cent of cases in this report), poor fusion and false retinal correspondence when it occurs early in life. (In 80 per cent of the authors' cases, convergent strabismus occurred before the age of 4 years.) There occurs, also, a more or less permanent impairment of the convergence and of the divergence function. Even though the amblyopia is relieved by complete occlusion of one eye, orthoptic training improves the fusion power and surgical correction results in approximate parallelism of the visual axes, spontaneous binocular fixation seldom results. It seems probable that convergent strabismus represents a much more profound pathologic entity than I was taught.

In some unknown way the convergence function seems to be linked with visual acuity, fusion and retinal correspondence. Consequently, in the majority of instances convergent strabismus represents a permanent impairment of the convergence and of the divergence function rather than a simple muscular anomaly. Because of the inability to reestablish binocular fixation, overcorrections and undercorrections must necessarily be numerous, especially when compared with those for anomalies of divergence.

The frequency of amblyopia sets up a problem which requires much patience and careful watching. I have advocated and practiced complete occlusion of the fixing eye, sometimes with success, and believe that long-continued occlusion is at times necessary. At other times I have practiced surgical correction of the deviating eye before normal vision was established in it, and subsequent occlusion in patients under 6 or 7 years of age may indicate congenital amblyopia.

Likewise, I have found a pronounced difference in individual responses to identical surgical procedures. In the few simple recessions performed, 8 degrees of correction was obtained from 4 mm. of recession. Spasm of the internal rectus muscle has disappeared after recession, but before operation I attempt to discount this finding as far as possible.

In spite of equality of vision, alternating strabismus apparently offers much more difficulty with surgical correction than the monocular type. I have frequently noted an up-shooting of the convergent eye. Often designated as a bilateral spasm of the inferior oblique muscles, I have classified the condition in many cases as double hyperphoria, finding that the hyperphoria stabilized itself after correction of the internal squint.

Because of variables, in all cases of more than 20 degrees or 40 D. of squint I have corrected approximately one half of the deviation on the amblyopic eye or the eye of greater deviation if the squint was alternating. After a few months, when the deviation if any has become stabilized, it is then corrected. In a few instances the anticipated second operation has been found unnecessary because of a satisfactory cosmetic result from the first operation or deemed unwise because of failure not due to technical difficulties. This procedure has enabled me to evaluate a repetition of the surgical method for the remaining deviation. The result of an operation on a normal fixing eye seems better than extensive resection and recession on an amblyopic eye.

The ideal age for operation must necessarily vary with the age of the patient at the onset of the strabismus, the type of strabismus, the

presence or absence of false correspondence and the fusion power. There seem to be, however, definite indications for operation. An amblyopic eye the vision of which is improved by constant occlusion of the fixing eye often fixes spontaneously when the deviation is corrected. I believe that occlusion is also indicated in the case of an amblyopic eye when fixation is difficult because of pronounced structural changes in the muscles. False retinal correspondence frequently disappeared spontaneously after operation at an early age, and operation would seem to be the best method of treatment for this anomaly. If there is any possibility of establishing some degree of fusion, an operation should be performed while the visual and motor systems are still in the process of development.

I believe the best management of convergent squint is definitely the same as that of the authors, i. e. operation before the adolescent age and when there is only 5 to 10 D. of uncorrected error. When there is poor fusion, immediate parallelism will probably not prevent divergence later. There is a tendency for the deviation to decrease rather than to increase for some time after the resection-recession type of operation.

The technic that I have employed for the past eight years is practically identical with that of the authors except for minor details. Resections and recessions are usually performed with buried catgut sutures and interrupted conjunctival sutures or a running suture. Believing that the external rectus muscles control divergence and the internal rectus muscles convergence, I have tried to adjust the correction on each muscle in accordance with the deviation for distance and for near vision, respectively. Whether this is of real value requires further trial and observation. Both eyes are uncovered on the fifth or sixth postoperative day, and no unusually severe reactions or complications have been observed.

I wish to congratulate the authors on their excellent presentation of this subject.

Dr. Frederick C. Cordes, San Francisco: About two years ago my associate, Dr. S. D. Aiken, suggested that as a substitute for recession the tendon be divided so that a large central band could be picked up, cut and allowed to recede, leaving narrow lateral bands intact. His suggestion was adopted, and since that time this method has been used extensively at the University of California and in my practice. We thought that it was an original idea until we learned that Chavasse in "Worth's Squint" (Worth, C. A.: Worth's Squint or the Binocular Reflexes and the Treatment of Strabismus edited by F. B. Chavasse, ed. 7, Philadelphia, P. Blakiston's Son & Co., 1939) described the procedure under the title of "Fenestrating Tenotomy."

In an attempt to evaluate the operation, a comparative study was made of a group of 120 cases taken from private practice so that in all the cases considered the surgical correction would have been done under similar circumstances by the same operator. This study brought out findings that correspond with some of those of Dr. Dunnington and Dr. Wheeler.

In reviewing these cases we also found that an operation, such as an O'Connor cinch operation or a recession, on one muscle had little effect unless something was done to the opponent muscle.

In cases included in the survey, an O'Connor operation of the usual type was done on the external rectus muscle, while various procedures were used on the internal rectus muscle. Of these, only the fenestrating tenotomy and the recession will be mentioned. With the fenestrating tenotomy 34 patients showed an average decrease of 32 prism diopters. The greatest correction was obtained when the recession was performed. With a 2 to 3 mm. recession, in 31 patients the average improvement was 35 prism diopters, while the 4 mm. recession gave an average decrease of 41 prism diopters, these figures corresponding rather closely to the figures given by the authors.

I found the results much more uniform when using the fenestrating tenotomy combined with the O'Connor cinch operation. This combination also has the advantage that neither muscle is completely detached from its original insertion and, therefore, the danger of a muscle's slipping postoperatively is avoided.

One point previously stressed by Dr. Dunnington bears repetition here, namely that a most important objective in all operations on muscles is accurate and tight closure of the conjunctiva. This precaution materially reduces the chance of infection and prevents the annoying granulomas so often seen when it is not observed. The closure of the conjunctiva with 0000 chromic catgut has also proved entirely satisfactory in my experience and does away with the necessity of removing conjunctival sutures postoperatively, which is a great help in dealing with small children. Since I have used this method of closure, conjunctival granulomas rarely occur.

The evaluation of surgical correction of strabismus can properly be done only when the case is studied in detail preoperatively and postoperatively in the manner followed by Dr. Dunnington and Dr. Wheeler. I feel that their paper is a valuable contribution to the subject of muscle surgery.

Dr. Maynard Wheeler, New York: I wish to thank Dr. Grant and Dr. Cordes for their discussions of this paper, and I regret that there is not time enough for more general discussion. We really expected quite a few comments.

I am glad that Dr. Grant stressed the matter of failure in obtaining binocular single vision when discussing the postoperative results. We have had the feeling that occlusion after operation might possibly upset the surgical result in some cases; so we have stressed preoperative occlusion and avoided postoperative treatment as much as possible.

We agree with Dr. Grant with regard to classifying as much as possible convergent and divergent anomalies into separate groups.

It is indeed interesting that Dr. Cordes gets comparable results from quite different procedures. This fact substantiates Dr. Dunnington's teaching that the exact procedure is much less important than a careful preoperative analysis and the use of the technic best understood by the operator. It is also our experience that combined shortening and weakening operations done at the same time give a greater effect than the identical procedures done at different times.

It should be emphasized that our results are determined from actual measurement of the deviation rather than on the basis of a cosmetic cure or a satisfied patient.

LAURENCE-MOON-BIEDL SYNDROME

REPORT OF A CASE IN A NEGRO FAMILY

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The Laurence-Moon-Biedl syndrome consists of retinitis pigmentosa, obesity, hypogenitalism, polydactylism and mental retardation. The literature on the condition up to 1939 has been collected by Cockayne, Krestin and Sorsby [1] (1935) and Sorsby, Avery and Cockayne [2] (1939), and among over 150 cases these authors found the disease reported only in the Caucasian race, with the exception of 2 cases among the Japanese and 1 from Egypt. It is therefore considered worth while to describe a case occurring in a Negro family (fig. 1).

The sibship consists of seven females and one male. The seventh child, Margaret P. (patient 1), showed four of the five parts of the

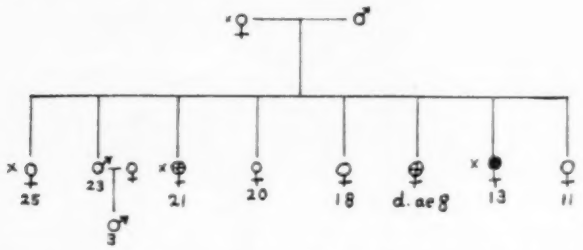

Fig. 1.—Pedigree of the P. family. The solid circle indicates the Laurence-Moon-Biedl syndrome; the circles containing a cross, related abnormalities, and *x*, that an examination was done.

Laurence-Moon-Biedl syndrome, having no evidence of hypogenitalism. In addition she had nystagmus and bilateral macular degeneration. The third child, Mary P. (patient 2), showed related abnormalities, consisting of bilateral macular degeneration and skeletal defects, not coming under the classification of the Laurence-Moon-Biedl syndrome. The third affected sibling was a girl who died at 8 years of age; she was reported by the mother to have had a sixth digit on each extremity and to have been remarkably obese. It was thought that she exceeded the family average in mentality. The physician in attendance at her death

From the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and University.

1. Cockayne, E. A.; Krestin, D., and Sorsby, A.: Quart. J. Med. **4**:93, 1935.
2. Sorsby, A.; Avery, H., and Cockayne, E. A.: Quart. J. Med. **8**:51, 1939.

stated that she died of "fat covering the heart." The remainder of the siblings were said to be unaffected, although only one was examined. As to the ascendants, the mother, who was examined, showed no ocular or gross skeletal defects. The father died at 76 years, presumably of senility, and it was stated that he was unaffected. None of the many aunts, uncles and cousins were known to be affected. There was no knowledge of consanguinity or of racial intermixture.

REPORT OF CASES

CASE 1.—*History.*—Margaret P., a 13 year old Negro girl, presented herself on Sept. 23, 1940 in the outpatient department of the Wilmer Ophthalmological Institute complaining of poor vision. Her sight had previously been "all right" except for difficulty in seeing at night. In the past two or three years she had sustained a decided loss of vision. In the same interval, from an alert and normally developed child she became unusually obese and mentally "slow." She had been known to have a double, fused right fifth toe. She had had only two menstrual periods, both in the past six months. The remainder of the medical history was noncontributory.

Fig. 2 (case 1).—Roentgenogram of the right foot, showing a supernumerary digit.

Physical Examination.—The patient was apprehensive and apparently mentally retarded, with symmetrically distributed obesity. She weighed 173 pounds (78 Kg.) and was 5 feet and 3 inches (160 cm.) tall. There were several superficial pustular lesions of the skin of the lower part of the legs. The right fifth toe was thickened and carried an extra nail, the supernumerary digit being fused to this toe. Gynecologic examination showed normal secondary sex characteristics and no evidence of hypogenitalism. The rest of the physical examination and the neurologic examination gave negative results.

Laboratory Data.—Urinalysis, tests of the blood serum and blood counts, including differential counts, revealed nothing unusual except eosinophilia with a count of 8 per cent. There was no evidence of sickling of the red cells. Roentgenograms showed a normal skull and sella. Roentgenograms of the right foot revealed a supernumerary fifth toe (fig. 2). The basal metabolic rate was — 8.

Ophthalmologic Examination.—There were no external abnormalities except a fine, rapid but inconstant nystagmus increased by efforts at fixation. The pupillary reactions, extraocular movements and tension as determined with the fingers were normal. Slit lamp examination disclosed that the corneas, anterior chambers and irises were normal. There were a few tiny, central, lamellar lens opacities, circularly arranged, but no posterior cortical changes. On examination of the fundi the media were seen to be perfectly clear. The disks were distorted and irregular in outline, but their margins were distinct and their color was normal. The arteries showed definite, uniform attenuation. The macular areas were the seat of severe degenerative changes; they had an irregularly stippled, shining, bronzed appearance and contained fine, scattered pigment deposits which assumed no consistent shape. The peripheral parts of the retina had a faintly tessellated

Fig. 3 (case 2).—Roentgenogram of the right hand, showing a short fourth metacarpal bone. The left hand was identical.

appearance, and there were many typical bone corpuscle–shaped pigment deposits having a centrifugal distribution. Vision was reduced to light perception in the right eye and to finger counting at 3 feet (90 cm.) in the left. Attempts at perimetry were discouraging, and at best one could be sure only of definite peripheral contraction.

CASE 2.—*History and Physical Examination.*—The only other living affected sibling was Mary P., aged 21 years. She had had normal vision up to the age of about 5 years; then in the course of two years her vision became reduced to its present status. There was an uncertain history of night blindness. The definite history of mental degeneration and rapid change in weight which was obtained in case 1 was lacking. The patient had had normal menstrual periods for several years. The general physical and the neurologic examination revealed only short fourth metacarpal bones bilaterally (fig. 3) and scattered, slightly raised, non-

ulcerated, lightly pigmented cutaneous lesions. The Wassermann reaction of the blood was negative. Roentgenograms showed generalized thickening of the bones of the skull, with no particular deformity of the sella.

Ophthalmologic Examination.—There was poor central fixation without true nystagmus. There were fine, dotlike, central nuclear lens opacities arranged in circular form, similar to those in case 1. Otherwise the anterior ocular structures were entirely normal. Ophthalmoscopically (fig. 4) the disks were sharply outlined and showed no pallor or cupping. The arteries were uniformly narrow. Choroidal vessels were not in evidence. The macular areas presented a granular, metallic appearance, fading off peripherally into normal retina. There were several scattered pigment granules in the macular areas, of which a few of the more outlying had suggestive bone corpuscle shapes. There were no pigmentary changes in the peripheral parts of the retinas. At the site of the fovea in each eye was a sharply outlined, irregular, dark brown area, homogeneous in character and not elevated. The vision in the right eye was 5/200 and in the left eye 2/200. In

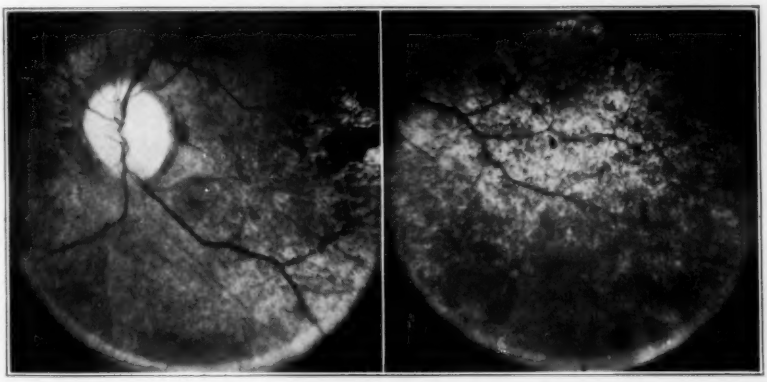

Fig. 4 (case 2).—The left fundus. There is extensive macular degeneration with some clumping of pigment. The arteries are narrow. The peripheral parts of the retina are normal.

this case, also, it was difficult to plot the peripheral or the central fields, but there was evidently nearly normal peripheral vision. This fact was evidenced by the patient's ability to get about by herself, while the first patient had to be led.

COMMENT

In 1866 Laurence and Moon [3] described the syndrome of retinitis pigmentosa, obesity, mental retardation and hypogenitalism. A fifth feature, polydactylism, was added independently by Bardet [4] in 1920 and by Biedl [5] in 1922. In spite of its long history, knowledge of the

3. Laurence, J. Z., and Moon, R. C.: Ophth. Rev. **2**:32, 1866.

4. Bardet, G.: Sur un syndrome d'obésité congénitale avec polydactylie et rétinite pigmentaire, Thesis, Paris, 1920.

5. Biedl, A.: Deutsche med. Wchnschr. **48**:1630, 1922. Raab, W.: Wien. Arch. f. inn. Med. **7**:443, 1924.

condition is limited to descriptive and genealogic observations. According to the data collected by Sorsby and Cockayne and their co-workers, the principal genealogic facts are that the condition appears for the most part in one generation of siblings, the ascendants and the descendants infrequently showing some of the components; the various features of the syndrome may be scattered among the members of a sibship, the males being slightly more often attacked; isolated cases are sometimes met with. It is confusing that the syndrome or parts of it often appear in conjunction with other ocular and skeletal defects, such as cranial deformities, cranial nerve palsies, muscular atony and congenital heart disease. Because of this admixture the process of disentangling possible variants from allied but different conditions has made little headway. Classification of these abnormalities and conclusions as to their mode of inheritance await a larger volume of observations.

SUMMARY

In a Negro sibship of seven females and one male, one female showed four of the five parts of the Laurence-Moon-Biedl syndrome, lacking hypogenitalism. In addition nystagmus and macular degeneration were present. Related ocular and skeletal defects were observed in a living sister and reported to have been present in a sister who was dead. None of the ascendants were known to be affected.

Wilmer Ophthalmological Institute.

OCULAR TORTICOLLIS—DIFFERENTIAL DIAGNOSIS

LOREN P. GUY, M.D.
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Mainly three specialties are concerned with the problem of torticollis. The orthopedic surgeon has to deal with torticollis caused by fibrosis of a sternocleidomastoid muscle (fig. 1) or by a defect in the cervical vertebrae (fig. 2).[1] The neurologist treats a variety of which the basis is either in a destructive lesion of the brain or in psychogenic factors caused by profound disturbances in the emotional life of the patient.[2] The ophthalmologist has to deal with a type of torticollis due to weakness of one of the oculomotor muscles concerned with elevation or depression [3] (fig. 3). Sometimes it is difficult to determine the type of torticollis with which one is dealing. Occasionally a patient suffering from torticollis of an ocular or neurologic nature receives orthopedic treatment, or vice versa. The literature on the subject is not particularly concerned with the differential diagnosis; so there is a need for discussion of the subject from a diagnostic viewpoint.[4]

The ophthalmologist would do well to approach the problem of diagnosis in a systematic way. The first step would be to get a complete history of the case. The date of onset is important. If the disorder seems to have been present from birth, it is well to inquire about the type of delivery, whether forceps was used or if there was a breech presentation.[5] If the torticollis came on after birth it is necessary to determine whether there was an accident. Mental maladjustment should be considered. The treatment the patient has received and the course of the disease are important factors. Often old photographs, especially snapshots, will be of help in eliciting the history.

Examination of the eyes should include a minimum of the following six tests: First, complete refraction with cycloplegia. Second, measurement of the oculomotor muscles in the primary position, that is with the

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Read before the Section on Ophthalmology at the Ninety-Second Annual Session of the American Medical Association, Cleveland, June 6, 1941.

1. Hough, G. D. N., Jr.: Surg., Gynec. & Obst. **58**:972-981 (June) 1934.

2. Alpers, B. J., and Drayer, C. S.: Am. J. M. Sc. **193**:378-384 (March) 1937.

3. White, J. W.: Am. J. Surg. **4**:77-79 (Jan.) 1928.

4. Lyle, T. K.: Tr. Ophth. Soc. U. Kingdom **56**:167-193, 1936.

5. Eddy, H. C.: Am. J. Surg. **17**:100-103 (July) 1932.

eyes looking straight ahead and fixed at 20 feet (610 cm.) and then at 13 to 17 inches (33 to 43 cm.). If glasses are needed, the measurement should be done while the patient is wearing his glasses as well as while he is not. Third, measurement of the oculomotor muscles in the six cardinal positions, that is eyes up and right, eyes right, eyes down and right, eyes up and left, eyes left and eyes down and left. This test is supplemented by noting the excursion of the eyes in these fields. Square prisms, as advocated by Duane and White, are used. Fourth, a test for convergence. Fifth, observance of the fixing eye for distance and for near vision. Sixth, a test for the presence and the degree of fusion. If no vertical oculomotor error is found, the eyes may be ruled out as a cause of torticollis unless ptosis of a lid is present.

Fig. 1.—Torticollis due to contraction and fibrosis of the left sternocleidomastoid muscle; note the tight muscle in the neck and the facial asymmetry.

In addition to these ophthalmic tests, the oculist may go further and note whether the patient has facial asymmetry and tightness of the sternocleidomastoid muscles with limitation of flexion and rotation of the head. He can determine whether the torticollis can be overcome voluntarily or whether it is fixed or spasmodic. A roentgenogram of the cervical portion of the spine should be taken as a means of discovering lesions in the vertebrae. The patient's nervous and emotional responses should be appraised. If all the foregoing procedures are followed, the torticollis in a particular case should be classified fairly readily. A summary of the differences between the three types of torticollis is presented in the accompanying table.

Theoretically ocular torticollis is dependent on some degree of single binocular vision. Hence, one would not expect to find it in the newborn;

it would not appear until fusion had developed. Torticollis in an infant is apt to be due to hematoma or fibrosis of a sternocleidomastoid muscle or to an injury to the cervical vertebrae. Such torticollis often appears to result from breech delivery, whereas ocular torticollis seems fre-

Fig. 2.—Torticollis due to fracture of the fifth cervical vertebra.

Fig. 3.—*A*, a patient with ocular torticollis with the head abnormally flexed and rotated; there is no facial asymmetry. *B*, the action of the right inferior oblique muscle, the cause of the torticollis. *C*, the carriage of the head after myomectomy of this muscle.

quently to result from forceps delivery.[5] Spasmodic torticollis occurs usually after the third decade of life.

A complete history of the case is always of paramount importance; for example, in the past six months I have observed a patient suffering from ocular torticollis which followed an injury to a trochlea of a superior oblique muscle and another patient whose ocular torticollis was due to trauma to a superior rectus muscle. Figure 2 illustrates torticollis due to fracture of a cervical vertebra.

As to a patient's mental attitude toward life as a factor in torticollis, it is well to remember that maladjustment may be a result of the deformitiy as well as related to the cause.

Refraction and measurement of the oculomotor muscles in the primary position at 20 feet and at 13 to 17 inches with and without glasses dis-

Six Important Differences in the Varieties of Torticollis

	Ocular	Orthopedic	Neurologic
1.	History of forceps delivery is frequent	History of breech delivery is frequent	History of normal birth is usual
2.	Torticollis is present since development of fusion, at about the age of 2 years, or since accident	Torticollis is present since birth or accident	Torticollis comes on usually after second or third decade
3.	Deformity can be voluntarily corrected by patient	Deformity is fixed	Deformity is intermittent
4.	Examination of ocular muscles shows a vertical error or ptosis of an upper lid	No vertical error of ocular muscles is present	No vertical error of ocular muscles is present
5.	There is no facial asymmetry	There is facial asymmetry if torticollis occurs before age of 16 to 18 years	There is no facial asymmetry
6.	Roentgenogram of cervical portion of spine is normal	Roentgenogram of cervical portion of spine may reveal deformity	Roentgenogram of cervical portion of spine is normal

closes the part accommodation may be playing in a muscle imbalance. Accommodative oculomotor disorders do not cause torticollis, but they may be associated with a nonaccommodative error that can be the basis.

The most important of all the ophthalmic tests for the diagnosis of ocular torticollis is measurement of the ocular muscles in the six cardinal positions. Square prisms are used. They are more easily handled than round prisms, and with practice adeptness is acquired. In the measurement of the muscles there are two important points which might be emphasized: First, one should make certain that the patient's head is held as straight as possible, and second, one should be on as nearly as possible the same level as the patient. Failure to observe these two points will cause one to make errors in measurement.

It is important to note which eye fixes. If the eye in which there is a paretic muscle fixes, the spasm is usually greater than if a normal eye fixes. As a result the torticollis may be expected to be greater. Surgical judgment is often influenced by this factor.

Fusion may be measured by many methods. Worth's amblyoscope is useful. Many elaborate modifications of this instrument are available. A less expensive instrument, which is nearly as satisfactory if used with prisms, is the Bishop-Harmon apparatus. An ordinary stereoscope can be used in most cases. Fusion is said to be the basic element in the pathogenesis of ocular torticollis. Without fusion, theoretically there would be no attempt to overcome diplopia by turning and twisting the head. Yet one occasionally sees a patient with ocular torticollis in whom fusion is difficult to demonstrate. How the development of fusion by ocular exercises can be used to overcome torticollis is difficult to understand, but one sees reports of this in the literature.[6]

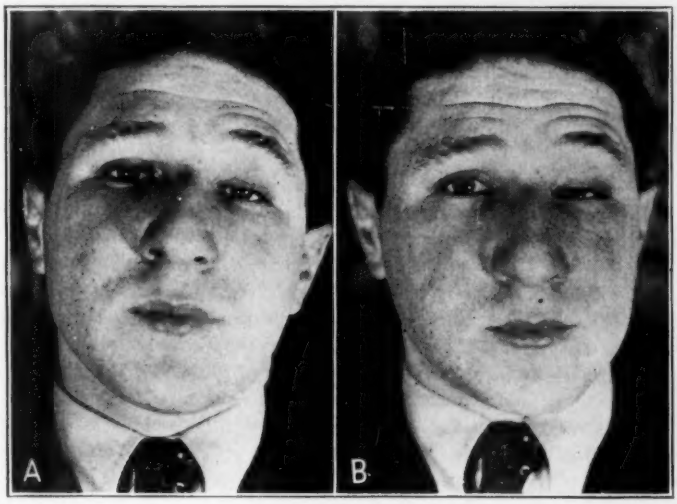

Fig. 4.—*A*, torticollis with abnormal flexion and rotation of the head due to ptosis of the left upper eyelid; the oculomotor muscles are normal. *B*, the patient voluntarily overcoming the torticollis; note the increase in the ptosis.

It is said that facial asymmetry results from ocular torticollis.[7] Many hundreds of patients were examined for whom especial attention was directed toward facial asymmetry, and in none could the torticollis be attributed solely to an oculomotor defect. Slight scoliosis is present sometimes in patients suffering from torticollis of ocular origin but not any more often than in patients without a vertical oculomotor error. Ocular torticollis can be overcome by the patient voluntarily, and during sleep it may not be present. As a result, the bones, muscles and nerves

6. Cass, E. B.: Proc. Roy. Soc. Med. **30**:1264-1268 (Aug.) 1937.

7. Bruce, G. M.: Am. Med. **40**:485-487 (Nov.) 1934. Levin, I. M.: Ocular Torticollis in Children, Am. J. Dis. Child. **44**:1026-1039 (Nov.) 1932.

of both sides of the body can develop normally and rigid postural deformities do not occur.

There is a type of torticollis of ocular origin that has not often been discussed, that due to ptosis of a lid. Figure 4 illustrates this type. It is not a diagnostic problem.

In a study of all types of torticollis in patients admitted to the New York Orthopaedic Dispensary and Hospital during the past five years, a large number were found to have had torticollis of ocular origin. It was interesting to note that nearly all these patients had previously been examined by ophthalmologists and had received no advice about the ocular basis for the postural defect. It seems justifiable to make the statement that ocular torticollis is more prevalent than physicians generally realize.

Dr. Benjamin Farrell and Dr. Alan DeForest Smith, of the New York Orthopedic Hospital, gave me their help in this study.

40 East Sixty-Second Street.

ABSTRACT OF DISCUSSION

Dr. A. D. Ruedemann, Cleveland: Ocular torticollis is a common finding in clinics where a complete examination of the eyes is part of the general examination. To be sure one must contest with the medical and the orthopedic specialist as to whom the patient should be referred. My patients with torticollis have always been referred to the orthopedic department, and I have removed from them the Thomas splint and have prescribed orthoptic exercises or operated on the vertical muscles and corrected them.

Dr. Guy is to be complemented for calling attention to torticollis that should be treated by the oculist. The oculist sees many patients with the acquired type of torticollis. One of the early lesions associated with thyroid disease is paralysis or weakness of the right superior rectus muscle. In about 30 per cent of the cases of muscle paralysis associated with thyroid disease the right superior rectus is the muscle involved, and therefore there is a great deal of acquired torticollis.

Patients with congenital torticollis do have facial asymmetry. Roentgenograms of the neck must be made in order to rule out cervical arthritis, which is a rather rare disease. Pain in the neck is a common sign of the acquired type of torticollis and is not uncommon in the congenital type, especially when the youngster becomes a student and attempts to do prolonged work.

I try to differentiate between mild pain in the neck on prolonged work, severe spasm in the neck, prolonged sharp pain and pain which the patient cannot tolerate, which sometimes becomes so severe that it simulates the pain of a brain tumor.

I should like to show a slide to bring out some of the points that were emphasized by Dr. Guy on the congenital type, and I shall show also some slides demonstrating the acquired type. (Slides were shown.)

One must take photographs and measurements of the neck in order to see exactly how much asymmetry of the neck one could get from twisting the head around in order to keep the eyes in position.

The neck muscles are eye muscles primarily. Their main function is to rotate the eyes in position and to get the nose out of the way so that one can look straight ahead at the object one wishes to see. Their function is to keep the head in a vertical and horizontal position to make seeing easy and comfortable. The

ocular muscles are the fine adjusters of vision, and if the ocular muscles do not put the eyes in position, then the neck muscles must attempt to do so. Asymmetry occurs when the neck attempts to do the job for the eye muscles.

In the congenital type, with tilting of the head in an effort to get the eyes in position to see, the patient has pain coming down the ligaments which cover the trapezius muscle and out over the shoulder when he attempts to straighten the head. He may have pain along the superior and the inferior nuchal line. The pain is deep seated and the muscles are seen to be hypertrophied if one examines the posterior part of the neck.

Pain at the tip of the mastoid is common with a vertical muscle error. Such pain may be caused by spasm of the sternocleidomastoid muscle. Any of the muscles concerned with the rotation of the head are posterior except this one, which attaches by two heads, the sternal and the clavicular.

The deep muscles of the neck are rotators of the head and fasten at the base of the skull, and deep pain, simulating pain or maladjustment of the atlas and axis, is frequently due to these deep muscles, which become irritated by the constant attempt to level the head.

The deep rotators of the head are posterior and fit around the foramen magnum. This patient (slide) had sternocleidomastoiditis and at the same time had a vertical muscle error. She was not made comfortable by an operation on the sternocleidomastoid muscle; an operation on the eye was necessary to enable her to carry on as a secretary.

This patient (slide) has facial asymmetry, and, as Dr. Guy brought out, in patients with spastic torticollis facial asymmetry is most common. She also has a relative hypertrophy of the right muscle.

Patients with vertical muscle errors due to previous endocrine disease do not have true paresis or persistent paresis, but rather muscle fatigue, and with prolonged use acquire their vertical errors. They really start out with a pain in the neck—they are a pain in the neck—and exercise will help them.

Persons who have merely vertical muscle weakness can be helped by orthoptic exercises.

This is another patient with muscle errors associated with acquired torticollis (slide). Such patients complain of pain, suboccipital pain, pain on prolonged use of their eyes.

To recapitulate, there is a large group of patients who have vertical muscle errors of the acquired type. They do not have facial asymmetry as an outstanding feature. The congenital type of torticollis may be associated with facial asymmetry, The main sign besides the phoria is pain in the neck, and this is not due to cervical arthritis.

DR. DON MARSHALL, Kalamazoo, Mich.: Torticollis is a condition that frequently is not recognized. Several of the patients reported on by Dr. Guy had previously been examined by prominent ophthalmologists, who failed to note the ocular basis for the torticollis. The majority of orthopedic textbooks and articles on this subject fail to give proper emphasis to the possible ocular basis, and often they omit even mention of this cause.

Torticollis can be caused by a large number of conditions. Commonly it is due to a short sternocleidomastoid muscle, resulting presumably from disease or damage occurring in utero or occasionally at birth. Rarely congenital torticollis is due to an anomalous vertebra or to a cervical rib. Acquired and transient torticollis may be due to (1) myositis resulting from infection in the pharynx or tonsils; (2) tuberculosis of the cervical portion of the spine; (3) dislocation, fracture or

arthritis of the cervical portion of the spine; (4) a paralytic or spastic lesion involving the spinal accessory nerve, and (5) tic or psychoneurosis.

A third and last type of torticollis is due to labyrinthine disease, cerebellar tumor, hemorrhage into the central ganglions or, finally, to ocular abnormalities. The ocular abnormalities include (1) paresis or spasm of the vertically acting ocular muscles, (2) nystagmus and (3) oblique astigmatism of considerable degree. It is obvious from this list that discovering the cause in a given case demands a broad view of the field rather than the narrow outlook of either the orthopedic or the ocular specialist.

Although a patient with frank torticollis does not often seek the ophthalmologist, it is surprising how many patients with ocular difficulties are seen to have some degree of head tilt if they are observed and studied for this condition. This group consists, of course, of persons with a vertical imbalance of the ocular muscles, and, from the viewpoint of diagnosis and therapy, except for the torticollis itself any patient with abnormal hyperphoria might be included.

Ocular torticollis has been defined as compensatory adjustment, tilt or position of the head to overcome a vertical diplopia. This definition would apply in the majority of cases, in which from spasm or paresis there is a vertical imbalance of the ocular muscles. More rarely, ocular torticollis has been found as an effort to improve visual acuity in cases of nystagmus and of high oblique astigmatism. In nystagmus the patient places his direction of gaze in the field of the shortest or slowest swing of his eyes. If the head is tilted to avoid diplopia, binocular vision must be present, and from this reasoning the conclusion has been reached that binocular vision is an essential component of ocular torticollis. However, White has shown that neuromuscular or proprioceptive sensation must also be a factor, since patients have been found with torticollis supposedly on an ocular basis in whom one eye was amblyopic or even blind, and the essayist has stated that it is often difficult to prove the presence of binocular vision.

The diagnosis of ocular torticollis depends on showing that there is hyperphoria or hypertropia which varies in the different directions of gaze and that this muscle imbalance is the cause of the head tilt rather than a structural or a functional defect in the muscles and nerves of the neck or in the spinal vertebrae. The usual patient has paresis of the superior rectus muscle on the side of the tilt, with overaction of the opposite inferior oblique muscle. Sometimes there is instead paresis of the opposite superior oblique muscle, and there is considerable difference of opinion as to which of the superior muscles is the usual offender.

To make the diagnosis a careful and complete examination of the ocular muscles after the taking of a complete history is essential. Much the same routine is used as in any case of vertical strabismus or phoria. In ocular torticollis the ocular muscles are not normal, there being hyophoria on the side of the tilt. The chin is directed down and toward the side of the tilt. The sternocleidomastoid muscle is generally normal, and the head can voluntarily and easily be put in the normal position. This may, however, cause ocular confusion or diplopia unless one eye is shut, and it was this fact which helped lead originally, years ago, to the discovery that wryneck might be due to eye trouble. Diplopia is greatest when the patient looks up and to the side of the tilt and least when he looks to the opposite side. When he looks to the side of the tilt, the opposite eye goes up and in.

In true torticollis, in contrast, the chin is up and away from the side of the tilt. The head cannot be put in the correct or an overcorrected position because of fibrous shortening of the sternocleidomastoid muscle. The movements of the ocular muscles and the balance are normal.

The question arises whether ocular torticollis long maintained may cause secondary structural changes in the shape and the development of the head, the muscles of the neck and even the spine. Dr. Guy finds that secondary changes remote from the eyes seldom occur, and he is in agreement with most, but not all, other observers. The explanation advanced seems logical, that, since with the eyes shut in sleep the head can and will assume a normal position, secondary changes do not get an uninterrupted opportunity to develop.

Can ocular torticollis be corrected by orthoptic exercises? The answer will not really be known until such exercises have been tried in a large series of cases. Theoretically correction would seem possible if, but only if, the vertical imbalance could be overcome by the exercises, and it is well known that vertical imbalance is a defect for which orthoptic exercises hold little hope. Therefore most observers agree that the treatment of a marked vertical defect, such as might cause torticollis, is surgical. Mention should not be omitted, however, of the fact that in certain adults with torticollis, particularly those with good binocular vision in the tilted position and those with variable binocular vision or with suppression of the vision of one eye, it is often better, or just as well, not to attempt to correct the condition. In the one case there may be difficulty in reeducating the neck muscles, and in the other treatment may be without benefit.

Dr. Thomas D. Allen, Chicago: I wish to report the case of a relative of mine, a woman 40 years of age, who was injured thirty-four years ago by falling from a horse and having her sister fall on top of her. The sister dug her elbow into the patient's left eye. Subsequently, the patient went around with her head tilted far over to the left side. She had no pain, no facial asymmetry and no refractive error, and no one paid any attention to her carriage until in the course of general ocular examinations of the family I noticed it. She was fitted with increasing prisms because the left inferior oblique muscle had been torn clear off, so that there was no upward movement of the eyes. After three or four changes, the total prism correction having reached about 12 prism diopters, a complete tonometry of the right superior rectus muscle was done, with resulting immediate tortional phoria; vertical and horizontal lines were oblique. This disappeared spontaneously within three weeks.

Dr. George Guibor, Chicago: May I call attention to the fact that torticollis exists in association with hyperphorias as well as hypertropias and that the vertical deviation is influenced by the torticollis? I have a slight head tilting produced by a left hyperphoria of only 3 prism diopters. When I am tested with the Maddox rod no hyperphoria is found if my head is tilted. Yet hyperphoria becomes manifest if my head is held erect.

Regarding treatment of patients with severe hypertropia (a secondary deviation) associated with hypotropia of the opposite eye caused by paresis of an elevator muscle, one may prescribe prisms base up before the lower eye and the secondary deviation (the hypertropia) will decrease in amount.

A second fact of interest is that in alternating hypertropia there may be alternating torticollis. Certain types of alternating hyperphoria are caused by supranuclear defects and not by a weakness in any one elevator muscle.

I should like to ask the essayist one question. What is the minimum vertical deviation that he has seen which has produced head tilting?

Dr. Loren Pritchard Guy, New York: Several interesting points came out in the discussion. One was which of the vertical muscles might be responsible.

It really doesn't matter. My experience, however, has been that the superior rectus muscles are by far the most often responsible.

I did not say much about therapy, but since the subject has been brought up I might say that the New York Eye and Ear Infirmary has an elaborate orthoptic department. Hundreds of patients have been treated, but the treatment of vertical errors does not seem to succeed. I have never noted a case that was properly controlled, in which measurement was done before and after the exercises, in which there was the slightest benefit from orthoptic exercises.

Dr. Guibor asked about the amount of error. I saw a patient—I think Dr. White saw her in consultation with me—a young woman who had a 10 or 15 degree tilt and had only 0.5 prism diopter of hyperphoria. It was corrected by prisms.

OCULAR CONDITIONS COMMON IN INDIA AND THEIR LOCAL TREATMENT WITH SULFANILAMIDE

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AND
M. N. MAITRA, M.B.
CALCUTTA, INDIA

Because of the finding of Rambo [1] that sulfanilamide applied locally in the eye, even when injected, did not produce any deleterious effect and because it was thought that the effective concentration and prolonged contact would be more easily obtained through use of the drug in the form of an ointment, this method was tried in the treatment of certain infective and noninfective conditions of the conjunctiva and cornea, e. g. trachoma, keratoconjunctivitis with adenitis and postoperative sepsis, and the sequelae of these conditions, such as xerosis and leukoma. The observations on trachoma will be published in another journal; the rest are presented herewith.

Because of the effect of the drug in the noninfective as well as in the infective conditions, a study was undertaken for determination of it effects on the normal conjunctiva and cornea and on aseptic wounds of the cornea. The experimental findings seemed to confirm the clinical observations, as will be shown later on.

KERATOCONJUNCTIVITIS WITH ADENITIS IN CALCUTTA

Keratoconjunctivitis with adenitis is somewhat akin to trachoma in the formation of pannus and papillary hypertrophy and the formation of granules in a certain percentage of cases. Pannus, although not an invariable accompaniment, may occur in the acute, subacute and chronic stages, and papillary hypertrophy appears in the subacute stages. Granules, however, are rare. As a rule the manifestations are acute. The disease of the cornea generally takes the form of keratitis, either superficial or deep, or ulceration, which may be one of various types. The most important feature distinguishing the disease from trachoma is the absence of formation of scar tissue either of the conjunctiva or

From Hemchandra Ophthalmic Institute and Jatindra Memorial Laboratory.

1. Rambo, V. C.: Effect of Sulfanilamide as Determined in the Eyes of Rabbits, Am. J. Ophth. **21**:739-746 (July) 1938.

of the cornea. In trachoma blindness supervenes mainly as a result of slow but relentless progressive fibrosis of the conjunctiva, cornea and lids, whereas in this disease loss of vision is brought about usually either by acute destruction of the cornea or, in cases with a favorable prognosis, by formation of leukomatous patches, with their inherent defects.

Bacteriologically, smears prepared with Giemsa stain show acidophilic coccoid bodies similar to those found in trachoma, but basophilic bodies are not seen. Histologically, the disease is more cellular-exudative than trachoma, which is a lymphocytic infectious granuloma with selective follicular localization and little reticulum or endothelium (Krückmann, 1932).

Sanyal's [2] conjunctivitis is distinguished clinically by the absence of yellow areas of degeneration, corneal complications and enlargement of the preauricular gland and bacteriologically by the absence of coccoid bodies.

Owing to deep penetration of the virus in the subepithelial layers of the conjunctiva and cornea, although the usual methods of treatment control the symptom complex it is not possible in many cases to eradicate the disease completely, because the commonly used drugs are unable to reach the deeper tissues. This is why on frequent occasions one has to resort to such remedies as milk protein, an aqueous solution of iodine introduced intravenously and chiniofon. Even then one is not always successful. The result is that many patients not only become carriers, infecting other persons, but themselves have acute exacerbations during seasonal changes. Nor are strong irritants, such as are used in the treatment of trachoma, suitable medications. This fact is evident from the following case report:

CASE 1.—D. L., a Hindu day laborer aged 30, came from the country to our clinic with pain in both eyes and a copious discharge. The condition had begun two weeks before as a mild attack of redness of the eyes with a slight discharge. It was gathered from his statements that he had been treated in the beginning with 5 per cent mild protein silver and 0.5 per cent yellow mercuric oxide ointment. These preparations practically cured his symptoms within a week, and he stopped the treatment. Within three or four days, however, he had another attack of acute symptoms, and he was given the same medicines. This time there was not much relief. Strong protein silver in concentrations as high as 4 per cent was then used. As this also failed, 15 per cent silver nitrate solution was painted on the everted eyelids each morning and 5 per cent copper sulfate in glycerine each evening. After two days the eyes became much more swollen, the pain increased and the discharge was copious.

2. Sanyal, S.: Keratoconjunctivitis with Adenitis in Calcutta, Am. J. Ophth. **19**:982-988 (Nov.) 1936. Duke-Elder, W. S.: Sanyal's Conjunctivitis, in Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1938, vol. 2, p. 1659.

Examination of the right eye disclosed considerable edema of the eyelids, which had to be separated with a speculum. The bulbar conjunctiva was chemotic and red and contained, irregularly distributed, numerous yellow-white phlyctenlike bodies of various shapes. There was papillary hypertrophy of the upper tarsal conjunctiva, and the lid could be everted only with much difficulty. The preauricular gland was enlarged and slightly painful. The cornea had a small infiltration on the upper one fourth, from 11 to 2 o'clock. The rest was normal. The left eye showed a similar picture, but the cornea was free from involvement. Vision was 6/9 in the right eye and 6/6 in the left.

Smear preparations revealed scanty bacteria in the epithelial cells and pus cells in all stages of degeneration. While we were securing scrapings, the nodules had a hard gritty feeling, and it was therefore decided to investigate them. With this end in view we snipped off two adjacent phlyctens and, after fixing

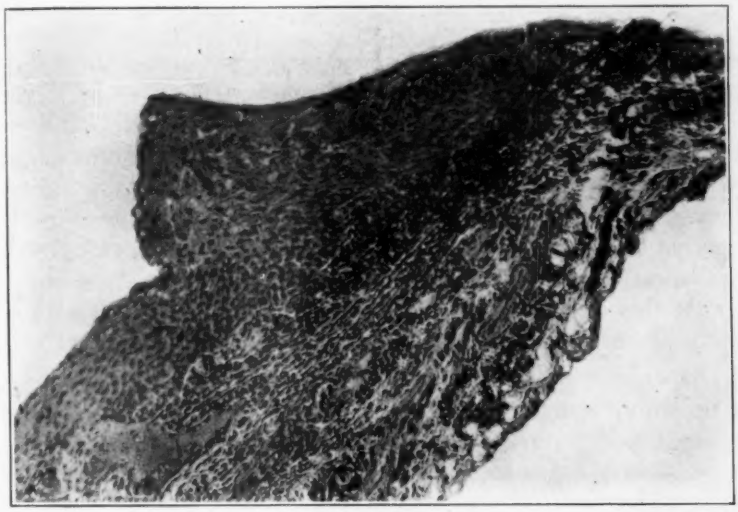

Fig. 1.—Keratoconjunctivitis with adenitis; section of the conjunctiva showing hillock-like hyperplasia of the epithelium and intense subepithelial infiltration after application of a strong caustic.

them in Bouin's fluid, made paraffin sections. Histologic studies showed that these nodules were mere hyperplasia of the epithelium which took a regular shape, like a hillock. The subepithelial layers showed dilatation of the capillaries, enormous proliferative activity of the endothelial cells and some edema. Apparently strong irritants are not well borne by the conjunctiva in acute stages, and it therefore tried to protect itself by hyperplasia (epitheliosis).

The eyes were washed with physiologic solution of sodium chloride and cold compresses applied three times a day for four days. Sterilized liquid petrolatum was then instilled twice a day. After four days the nodules disappeared, and the patient made an uneventful recovery within ten days.

In view of the difficulties in the treatment of keratoconjunctivitis, it was decided to use a 5 per cent ointment of sulfanilamide. Previous

to the application of this ointment, moist compresses were applied and physiologic solution of sodium chloride used for irrigation twice a day.

The following conclusions are based on the observation of nearly 500 cases in which this method of treatment was employed:

1. The relief of subjective symptoms begins within an hour of the first application of the drug and continues for twelve to eighteen hours after each application.

2. Characteristic flat yellow areas of degeneration on the palpebral and the bulbar conjunctiva may be removed within twenty-four to seventy-two hours depending on their size, especially their depth.

3. The discharge increases for the first forty-eight hours and then begins to diminish, continuing slightly until the termination of the disease (seven to twelve days).

4. Twenty-four hours after the first application of the drug, a smear preparation of scrapings from the tarsal conjunctiva shows an increased number of leukocytes, mostly in a state of degeneration. The majority of the epithelial cells, too, are in various stages of degeneration, showing a fainter staining reaction, and some are loaded with bacteria up to the nucleus. After seventy-two hours the epithelial cells stain more brightly; the coccoid bodies are seen mostly at the periphery of the cells. Within a week most of the leukocytes are healthy; so also are the epithelial cells. At this stage the patient is free from subjective symptoms. In about twelve days the smear is completely bacteria free.

5. Superficial infiltrations at the periphery of the cornea sometimes clear up within four or five hours; those at the central area take more time, about a week or ten days.

6. Superficial marginal ulcers heal within a day or two.

7. In deeper central ulcers, edema of the cornea diminishes and the infiltration becomes more localized to the ulcerated area within twenty-four hours. The ulcer takes ten to fifteen days to heal, according to its depth and extent. Ciliary injection, attendant pain, photophobia and lacrimation begin to subside within twenty-four hours.

8. Deeper keratitis takes three weeks to a month to clear up. When it reaches the posterior layers and is of long standing it may not disappear completely.

CASE 2.—*Acute keratitis.*

B. D., a woman aged 25, complained of a gritty sensation, photophobia, lacrimation and a slight discharge in the right eye. The condition had been present for five years with acute exacerbations during rainy seasons. Examination revealed moderate edema of the lids, and enlargement of the preauricular gland to about 1 cm. accompanied with slight pain. The conjunctiva of the upper lid presented a velvety appearance and was studded with yellowish, flat, irregular areas about 1 to 3 mm. in diameter. Each of these was surrounded by a small

blood vessel but was not itself secondarily vascularized. The lower lid also showed uniform congestion. The congestion of the bulbar conjunctiva extended nearly to the cornea. Laterally there were two yellow areas, each about 1 mm. in diameter and about 1 cm. from the cornea. The cornea looked dull, and the discharge was slight. Scrapings of the tarsal conjunctiva after being stained by Giemsa's method showed a moderate amount of leukocytes, all in a state of degeneration. Most of the epithelial cells were loaded with acidophilic bodies and were in various stages of disintegration. Many ghost cells were present. One day after treatment was instituted the subjective symptoms were at their minimum. The patient could open the eye with much more comfort, but the lids felt heavier. Both the upper and the lower lid were slightly more edematous. In the tarsal conjunctiva there was general hyperemia, and many of the smaller yellow areas had disappeared. No yellow spot was seen on the bulbar conjunctiva, and the hyperemia there had lessened. The cornea was clear.

Examination by smear showed an increased number of leukocytes per field, the majority of the cells being in a state of degeneration. The epithelial cells showed a slightly better staining reaction. The cocci now occupied chiefly the distant half. A few ghost cells were still present. Two days later no discomfort was present except a little heaviness of the lids and an occasional gritty sensation with slight lacrimation. There was practically no photophobia. On the tarsal conjunctiva the yellow areas, except a few on the outer side, had disappeared. Hyperemia and edema were still present. The bulbar conjunctiva was almost clear. A smear showed some of the leukocytes in a healthier condition. In many of the epithelial cells the cocci were near the periphery, which seemed to be slightly swollen. After six days of the usual application the photophobia was almost under control. The heaviness of the lids persisted; yellow areas could not be seen, and the bulbar conjunctiva was clear. Examination by smear revealed a diminution in the number of leukocytes per field. Many of these were healthy. Epithelial cells still showed mild infection; many of the cells stained better now.

On the tenth day the conjunctiva of the lids was slightly hyperemic, and slight heaviness was felt. Most of the leukocytes appeared normal. A few epithelial cells still showed the bacteria near the periphery. The number of these gradually decreased, until after five days more the conjunctiva was bacteriologically free. The edema of the lids persisted, and zinc alum lotion was prescribed, while the sulfanilamide ointment was discontinued.

CASE 3.—*Keratitic marginal infiltrates.*

M. L., a Hindu woman aged 50, had had intense pain in the forehead, photophobia, lacrimation and a slight discharge in the right eye for about a week. There was moderate edema of the lids, and the tarsal conjunctiva had a slightly velvety appearance, with yellow spots varying in size from 2 to 4 mm. in diameter at the outer side. The preauricular gland was slightly enlarged. On the cornea there were four grayish spots 2 mm. in diameter. These were slightly raised, were located near the periphery between 11 and 6 o'clock and were arranged concentrically with the margin of the cornea and about 3 mm. from it. The surrounding cornea looked slightly hazy. There was also a superficial ulceration about 3 mm. in diameter at 6 o'clock nearly 2 mm. from the limbus. The pupil was 3 mm. in diameter and its reaction sluggish. The tension seemed normal by palpation.

The left eye was blind from chronic glaucoma.

The ointment was applied at 3 p. m. the day of admission. At 7 p. m. the infiltrations at the upper margin had practically disappeared; only faint white areas marked their former position. The margins of the ulcer stained less brightly with methylthionine chloride U. S. P. (methylene blue). The subjective symptoms were much relieved. The conjunctival discharge and the edema of the lids were slightly increased. Next day the subjective symptoms were almost completely relieved. The cornea was clear at its upper half, and the ulcer was about 1 mm. in diameter. Two of the yellow areas had disappeared, while the conjunctiva remained velvety. The discharge was almost the same. The edema of the lids was still present, and the preauricular gland was still enlarged.

Two days afterward the cornea was clear except for whitish scar tissue at the position of the ulcer. The yellow spots were still present, and the preauricular gland was just palpable.

On the seventh day the cornea was clear; no scar tissue could be seen. The yellow spots had disappeared. A little edema of the lids persisted, with a slight discharge, while the conjunctiva was velvety. The ointment was discontinued after four days, when the conjunctiva was free from bacteria, and zinc alum lotion was prescribed in its stead.

PANNUS

Although pannus is not an invariable accompaniment of keratoconjunctivitis, the various types that are met with in association with trachoma are also seen in this disease. In a certain percentage of cases of keratoconjunctivitis accompanied with pannus eosinophilic cells may be found in the cornea itself. Since no allergen can be found, this fact suggests that a hypersensitiveness to the coccoid bodies develops in the cornea itself. The pannus is, however, amenable to treatment with sulfanilamide, as is that associated with trachoma. Experimentally, it has been found that instillation of a filtrate of the conjunctival scrapings into the conjunctival sac of the rabbit followed by light scarification of the cornea seems to produce pannus. But this condition requires further study, and a detailed report will be given later.

INTERSTITIAL KERATITIS

Clinically, two types of keratitis are seen, one vascular and the other avascular. In the vascular type, the blood vessels that enter the cornea are of the conjunctival type and therefore superficial. These vessels generally course through one-third to one-half the cornea and are then lost. Sometimes they collect in the upper part near the limbus and look like a salmon patch. Occasionally the cornea has only one or two blood vessels in the lower part, and these may be so fine that they are visible only through a loupe or with a slit lamp. The whole of the cornea is deeply infiltrated in irregular patches of varying thickness, some of which extend the entire depth of the cornea. There is some ciliary injection, and there may be associated iritis.

In the nonvascular type, which is somewhat more uncommon, there is a ground glass appearance of the cornea, in which there are no blood vessels. Under the slit lamp the infiltrations are seen to be confined

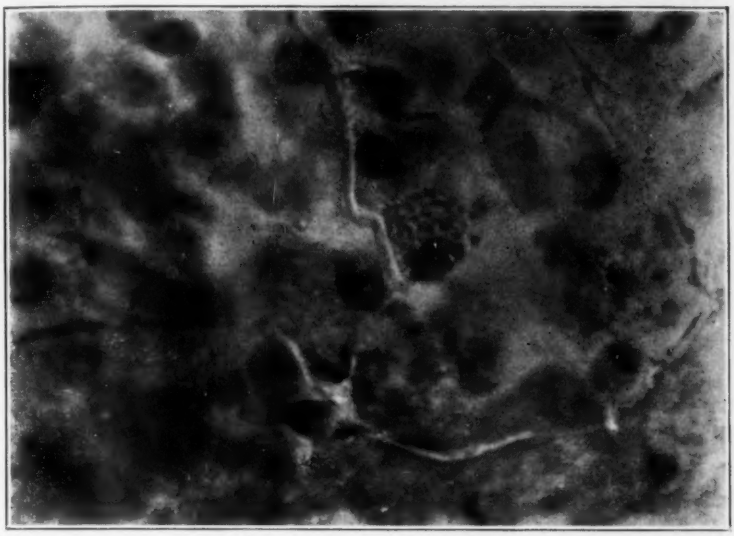

Fig. 2.—Pannus; eosinophilic cells and strands of fibrous tissue in the cornea.

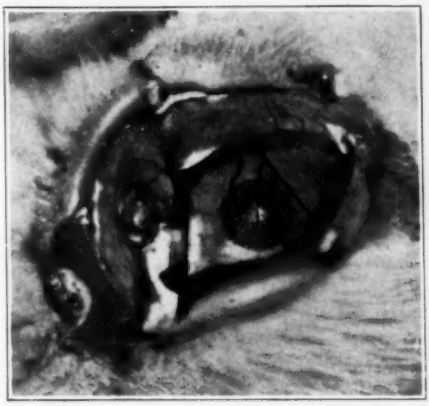

Fig. 3.—Experimental pannus in a rabbit (slightly retouched).

chiefly to the anterior layers, and they are of varying densities. No coincident iritis has been noted.

In the nonvascular type no hypersensitiveness to any substance is found, and in both types the reaction to tuberculin and the Wassermann reaction are negative.

The nonvascular type if untreated persists for months, with periods of quiescence and exacerbations like those of pannus. The vascular type shows a spontaneous tendency to clear up after a few months.

The vascular type of interstitial keratitis is somewhat similar to pannus in its histologic aspects. The avascular type, however, has some peculiarities:

1. The epithelium is either normal or shows irregular hyperplasia, which has (*a*) knoblike projections with bifurcations in one or two places, (*b*) a downward formation of papillae, (*c*) partial degeneration in places and (*d*) hyperactivity of the basal cells which causes a heaping up of the basal cuboid cells to the surface.

2. Bowman's membrane shows: (*a*) normal portions and (*b*) slight thickening in places with projections into the hyperplastic epithelial tissues which divide it into compartments and taper toward the surface, stopping about midway.

3. The substantia propria is characterized by edema, hyperactivity of the corneal corpuscles, dilatation of the lymph spaces, lymphocytic infiltration and the appearance of a few epithelioid and giant cells. In some specimens moderate infiltration with eosinophilic cells and partial necrosis are found. The edema is especially severe under Bowman's membrane, where the lymphatics, markedly dilated, are interspersed with edematous areas of various sizes, so that some distance beneath the membrane the corneal substance looks like a sieve and the epithelium is separated from the substantia propria. In the deeper layers, also, a little edema and irregular dilatation of the lymphatics are present. The deeper edematous areas are characterized by intense lymphocytic infiltrations with little corneal substance, and strands of fine fibers are seen running through them. These infiltrated areas generally take a more or less round or oval shape and are usually confined to the anterior parts.

CASE 4.—*Avascular keratitis.*

S. L., a Hindu boy aged 15, had come to our clinic because of complete loss of vision in the right eye together with photophobia, lacrimation and slight pain in the eyes. About two months before he had had an acute attack of redness of the eyes accompanied by some pain, dread of light, lacrimation and discharge. With medical treatment the symptoms subsided, but he was not completely cured. About fifteen days before he had again had an attack of redness of the eyes, with slight pain and photophobia. The discharge was slight. Within a day or two he began to notice some diminution in sight in the right eye, and this began to increase gradually. When he came to our clinic he had lost his vision completely, only perception of light and of its projection being present.

Examination revealed that the lids of the right eye were somewhat swollen and that the palpebral conjunctiva was velvety, with yellow dots on the outer side of the conjunctiva of the upper lid. The bulbar conjunctiva was almost normal. The preauricular gland was slightly enlarged. The cornea presented a ground glass appearance; no vascularization was seen. There was slight ciliary

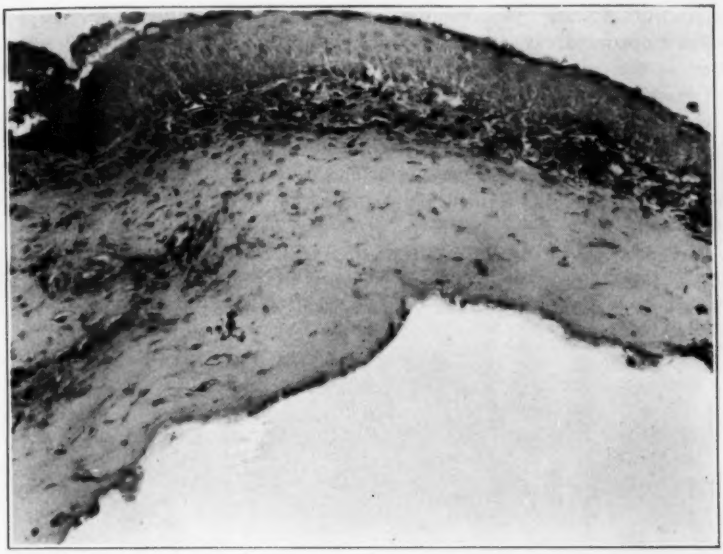

Fig. 4.—Interstitial keratitis in keratoconjunctivitis; section of the cornea showing hyperplasia of the corneal epithelium and lymphocytic infiltration just beneath Bowman's membrane. The corneal substance is comparatively free.

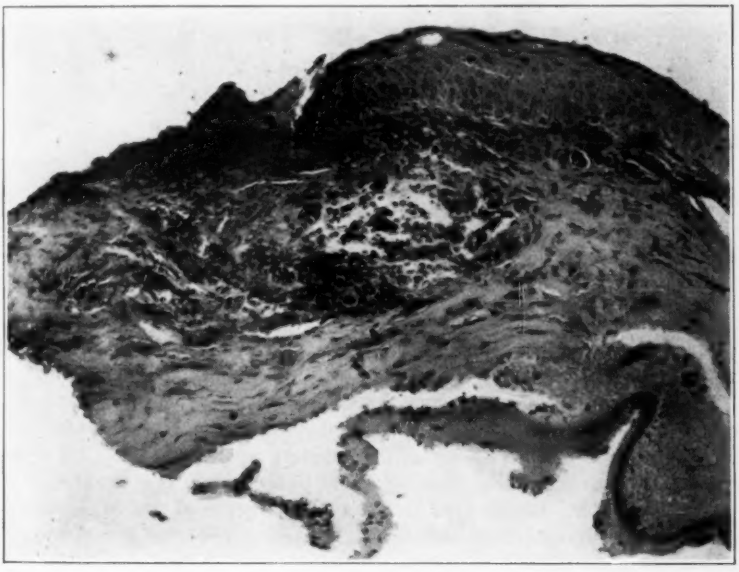

Fig. 5.—Interstitial keratitis; section of the cornea showing a localized infiltration and edema.

injection. Slit lamp examination revealed some irregularity of the surface of the epithelium. Scattered edematous areas in the subepithelial layers together with dense opaque areas here and there were present. The opacities were mostly confined to the anterior half. No deposit on Descemet's membrane was visible. Blood vessels were absent in the substantia propria. The iris reacted to light and was hyperemic. The Wassermann and the tuberculin test gave negative results. Scrapings from the conjunctiva stained by the Giemsa method showed the characteristic acidophilic coccoid bodies.

On June 7, 1938 the patient was advised to apply 5 per cent sulfanilamide ointment twice daily. On June 8 the ground glass appearance had been replaced by scattered areas of deeper infiltration separated by fainter superficial areas. The vision was perception of hand movements at 1 foot (30 cm.). The conjunctiva was hyperemic.

Fig. 6.—Interstitial keratitis; section of the cornea showing the presence of eosinophilic cells.

On June 10 the iris was faintly visible in places at the periphery. There was a general thinning of the opacity throughout the cornea. The vision was perception of finger movements at 4 feet (122 cm).

On June 12 a peripheral portion between 9 and 1 o'clock about 3 mm. in breadth was almost clear and vision was counting of fingers at 4 feet.

On June 14 the central opacity at the pupillary area seemed to be the densest, although there was a general thinning of the infiltration. The lower and lateral one third was clear. In the area between 1 and 5 o'clock the uniform opacity was replaced by dots of various sizes in between which the cornea was clear. There was a faint general haziness over other parts of the cornea.

On June 19 the central portion was also much clearer. The opacity now occupied an area 2 mm. in diameter near the center, and the pupil was visible.

The other parts except the medial portion were clear. In the medial area a few white dots considerably smaller than they had been were present.

On June 21 the whole of the cornea was clear except for a faint nebula in the center. The vision was 6/6.

CASE 5.—*Vascular keratitis.*

B. T., a Hindu woman aged 40, came to our clinic on Dec. 28, 1938 complaining of a dull aching pain in the right eye and in the forehead and photophobia, lacrimation and complete loss of vision in the right eye. The history was that eight months before she had had an acute attack of redness of the eye with lacrimation, photophobia, a gritty sensation and a discharge which continued for seven days. From the seventh day the pain in the eye increased and she began to perceive some dimness in her vision. Within a week her sight became reduced to perception of light. At this stage she went to a hospital, where the blood was examined, local remedies were applied to the eye, injections were made into the buttocks twice a week for one month and internal medicines were given. Although the pain in the eye diminished, the vision was not restored.

Examination revealed that the lids were slightly edematous. The conjunctiva of the upper and of the lower lid were slightly velvety. Small yellow areas characteristic of keratoconjunctivitis were seen on the outer side of the upper tarsal conjunctiva. The preauricular gland was slightly enlarged, but it was not painful. The whole of the cornea was deeply infiltrated, the depth varying. Two conjunctival blood vessels were seen, one at 1 o'clock and the other at 7 o'clock, proceeding nearly to the center. A few deep vessels could be detected. Ciliary injection of moderate intensity was present. The anterior chamber was not visible. The vision was reduced to light perception and projection.

The sulfanilamide ointment was applied, and within twenty-four hours the pain in the eye was much relieved. Clearing of the cornea commencing at the periphery was noticeable. Within a week there was a general thinning of the opacity over the whole of the cornea except an area 3 mm. in diameter in the middle. The clearing at the periphery was the most marked. Through the clearer portions of the cornea an old iritis with exudation in the pupillary area was visible. The pain was now negligible. The following week the more superficial areas of infiltration disappeared; small islets of deeper infiltration interspersed with small dots persisted. The central portion of the infiltration had become much thinner. The blood vessels were still present. Six days later the whole of the cornea was practically clear, but some scattered pinpoint areas remained, especially in the central portion. These did not show further improvement with sulfanilamide ointment, but use of the ointment was nevertheless continued for a week longer. Then atropine sulfate, ethylmorphine hydrochloride and yellow mercuric oxide ointment were substituted. The cornea took about twenty-one days more to clear completely. In the meantime there was some improvement in the iritis, and the vision came up to 6/36.

The patient was not heard from afterward.

DACRYOCYSTITIS

As a complication of keratoconjunctivitis the lacrimal sac is sometimes infected secondarily. Pathologically the inflammation differs somewhat from other types of chronic dacryocystitis, and smears prepared from discharge obtained by applying pressure to the sac, as well as from

scrapings of the epithelium obtained after removal of the sac, show acidophilic coccoid bodies which are characteristic of the parent disease.

Histologic study revealed the following changes in the sac:

1. In the epithelium are observed (*a*) irregular hyperplasia causing either a rise above the surface or a dip below the normal level, (*b*) a change in the character of the epithelium from cylindric to squamous and (*c*) partial degeneration at places.

2. In the subepithelial layer are observed (*a*) an infiltration of lymphocytes, plasma cells, endothelial cells and a few leukocytes, which may aggregate into follicles with ill defined capsules either immediately below the epithelium or at a certain distance from it, (*b*) a fibroblastic reaction confined chiefly to the inner segment, where irregular strands

Fig. 7.—Dacryocystitis in keratoconjunctivitis; smear preparation showing the coccoid bodies (Giemsa stain; × 1,800).

of fibrous tissues may be seen running either parallel to the epithelium or at right angles to it and (*c*) edematous areas irregularly placed.

The sac may act as a reservoir to contain pathogenic bacteria without itself being infected.

In the early stages, when the changes are not advanced, this condition is amenable to treatment with sulfanilamide. The technic of application was as follows:

> The affected sac was first pressed on through the skin to bring out as much of the detritus as possible, which was wiped away. A few drops of a 0.5 per cent sulfanilamide solution was then put into the sac. This expression and instillation were carried out three times at intervals of two to five minutes. A hot compress was then applied. Finally, the sulfanilamide ointment was applied in the conjunctival sac. The entire process was repeated two times a day.

Case 6.—*Keratoconjunctivitis with dacryocystitis.*

A Hindu man aged 35, seen May 15, 1939, complained of epiphora in the right eye for the past four and one-half years, with a slight discharge. Moderately

severe inflammation of the lacrimal sac was found. With pressure on the sac, a whitish mucopurulent discharge came out through the punctum. A smear of the discharge revealed degenerated leukocytes, staphylococci and acidophilic coccoid bodies, some within and some outside the epithelial cells, most of which were degenerated.

After the technic described was carried out for twenty-four hours the discharge increased and began to thicken; the sac became a little fuller. Three days afterward the discharge diminished and became much thinner, but the fulness of the sac persisted, while the epiphora diminished. After seven days the discharge was scanty and a smear showed most of the leukocytes healthy. The epithelial cells were much healthier than they had been, and bacteria were few. Four days later there was little or no discharge on pressure. A smear showed the leukocytes and epithelial cells to be perfectly healthy, and there were no

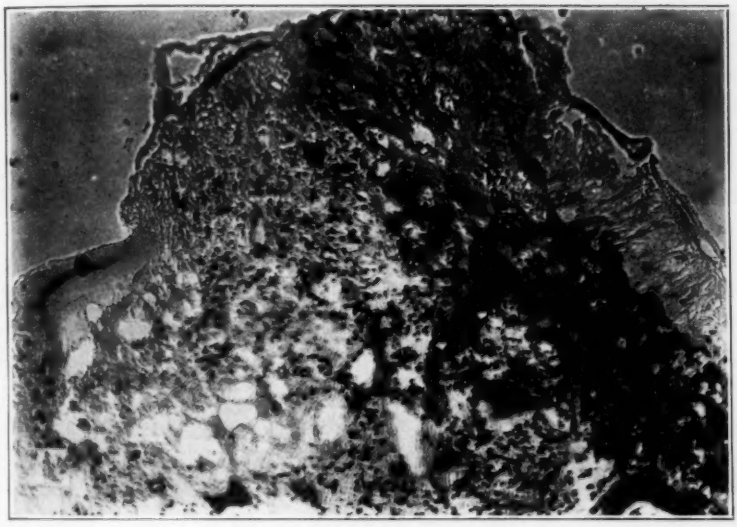

Fig. 8.—Dacryocystitis; section of sac showing fibrous tissue confined chiefly to the subepithelial layer (iron-hematoxylin stain).

bacteria. The sulfanilamide solution and ointment were now discontinued, and the following lotion was instilled into the conjunctival sac, the same technic being followed: 1 grain (0.06 Gm.) of albargin[3] in 1 ounce (30 cc.) of distilled water. After seven days there was no discharge and no fulness. The patient was completely relieved of epiphora.

POSTOPERATIVE SEPSIS

Postoperative infection of wounds has always been a source of anxiety to ophthalmic surgeons, although modern methods of asepsis have reduced its frequency almost to rarity. Four cases, however, of involvement of moderate severity were observed within forty-eight hours of

3. A preparation containing silver combined with a gelatose soluble in hot and in cold water.

infection. Sulfanilamide ointment was used three times a day in all these cases after application of a hot compress for ten minutes and irrigation of the eye with physiologic solution of sodium chloride. The eyes were not bandaged. In a case of infection following a corneal section a solution of 1 per cent atropine sulfate and solutions of ethylmorphine hydrochloride in increasing concentration beginning at 2 per cent were used, and the following results were noted:

Within twenty-fours the edema of the lips of the wounds began to subside. The general haziness of the cornea became more localized to the lips of the wound, and there was less congestion of the ciliary vessels. The exudation in the anterior chamber began to diminish within seventy-two hours. Within about the same time the infiltration of the cornea became confined to the margin of the wound, and the lips of the wound became flat. The exudation in the anterior chamber took three or four days more to be absorbed completely, and during this period the cornea returned to normal clinically. Administration of the drug was continued for about a week more as a precautionary measure.

In cases of flap infection the yellowish nodules became flatter and the edema of the conjunctiva more localized, and within seventy-two hours the clinical appearance became normal except for a little edema at the sites of the nodules.

It was interesting to note that the intense pain which is inherent in such infections was greatly relieved after the first application of the drug, so that no analgesic was required throughout the course of the treatment.

CASE 7.—*Flap infection.*

P. S., a Hindu woman aged 25, was first seen on Feb. 15, 1939 because of a septic infection of the wound five days after a trephine operation on the left eye. Examination showed the eyelids to be moderately swollen and the bulbar conjunctiva to be intensely congested and slightly chemotic. There was some discharge. On the outer side about 1 mm. from the trephine hole there was a golden yellow nodule about 1 mm. in diameter under the conjunctival flap. The surrounding conjunctiva for about 5 mm. was raised above the surface. The patient complained of pain in the eye and the head. Use of sulfanilamide ointment three times a day after application of a hot compress and irrigation with physiologic solution of sodium chloride was advised.

On February 16 the nodule was flatter, the general chemosis had disappeared and the patient could open her eyes more freely. There was some increase in the discharge. The pain was much relieved after the first application of the drug.

On February 18 the nodule was not noticeable but its place was marked by localized redness and edema. The edema of the lids had practically disappeared. The discharge was greatly diminished.

On February 2 the edematous area had become flatter and the congestion of the bulbar conjunctiva had practically disappeared. There was no discharge and no pain. On February 23 there was only slight localized congestion at the site of the nodule, and on February 25 the eye was normal.

CASE 8.—*Infection after corneal section.*

B. S., a Hindu woman aged 60, was operated on for cataract on Jan. 1, 1939. A sclerocorneal section was done. There was a slight prolapse of the iris, and the anterior chamber failed to reform until January 27. On January 29 infection of the corneal wound was noted, with the following picture:

The lips of the wound at 11 to 1 o'clock were swollen and were a dirty yellowish color. There was general infiltration of the whole cornea, with injection of the ciliary vessels. The bulbar conjunctiva was congested, and there was some discharge. Slight exudation in the anterior chamber was visible. The patient complained of neuralgic pain in the head.

Sulfanilamide ointment was used after application of a hot compress and irrigation with sterile physiologic solution of sodium chloride. This treatment was carried out three times a day.

On January 1 the infiltration of the cornea had become more localized to the lips of the wound, and these were flatter. The yellowish color had been replaced by a dirty gray. The exudation in the anterior chamber had greatly diminished. The pain was much relieved. The other conditions remained the same.

On February 2 the infiltration of the cornea was confined to an area about 3 mm. broad at the lips of the wound, which were now in apposition. There was still slight exudation in the anterior chamber.

On February 4 no dulness of the cornea was present. Repair of the wound was seen to be complete. The anterior chamber was free from exudation. There was absolute freedom from pain. Slight injection of the ciliary vessels was seen, and some hyperemia of the palpebral conjunctiva persisted. Continued use of the ointment for about a week was advised, and the patient was discharged as cured.

On March 10 the vision was 5/60 without correction and 5/9 with a + 11.00 D. sphere.

NONINFECTIVE CONDITIONS

Sulfanilamide is applicable not only in the treatment of septic conditions. There are certain noninflammatory conditions for which it has proved of some value. Of these, two may at present be mentioned, xerosis and leukoma.

XEROSIS

A. *Parenchymatous Xerosis.*—Of the various types of xerosis, parenchymatous xerosis, induced by trachoma, is the most difficult to deal with. When this condition is extremely severe, in addition to the changes in the conjunctiva there is a dry wrinkled condition of the corneal epithelium together with cellular infiltration in the substantia propria and pannus leading to loss of vision. Such palliative measures as putting in a drop of honey or some oily substance help to a certain extent.

Sulfanilamide is of benefit, especially in the early stages, in removing the dry, lusterless condition of the cornea and restoring the healthy condition of the conjunctiva. When small areas like Bitot spots appear on the cornea, with slight infiltration in the substantia propria, and the cornea looks dry and slightly scaly without much epithelial change, distinct improvement in the condition and therefore in vision may be

obtained through local application of the drug, the period of which depends on the density of the opacity and the changes in the corneal epithelium.

Even in cases in which the xerosis is extremely severe, in which the cornea has become rough and densely infiltrated, pannus has invaded from all sides and the conjunctiva is thick, leathery and epidermoid and thrown into folds, some regeneration may be induced, the amount depending on the restoration and the maintenance of the secretory activity of the conjunctival glands and the freeing of the lacrimal ducts. But the difficulties in these cases are twofold: 1. The drug, if administered for a long period, seems to lose its strength of action gradually. 2. If the drug is discontinued the condition may slowly develop again, the rate depending on the state of the secretory glands at the time the drug is discontinued, that is on how far the glands are able to take up their normal function. This difficulty, however, can to a certain extent be overcome by careful attention to the gastrointestinal tract and liberal administration of vitamin A.

B. *Epithelial Xerosis.*—As epithelial xerosis is a part of general degenerative changes in the ectodermal structures of the whole body due to a deficiency of vitamin A, it can be checked or prevented to a great extent by a diet rich in that substance. But the pathologic changes already induced by the deficiency are not generally removable through dietetic treatment alone. Local application of sulfanilamide is often of distinct benefit in inducing a healthy condition of the tissues provided the body has enough of vitamin A at the time the treatment is instituted.

CASE 9.—*Parenchymatous xerosis.*

A. B., a Hindu man aged 32, gave a history of dimness of vision, with dryness and a gritty sensation in both eyes for the last eleven years. Lacrimation was absent from both eyes. Fifteen years earlier he had contracted trachoma, and he had been treated for about a year with 2 per cent silver nitrate solution and ammoniated copper solution lapis divinus in the usual way. During the course of treatment pannus and corneal ulcers developed and the vision began to fail. Three years later iridectomy was done in both eyes, but vision did not improve much.

Examination of the right eye revealed that the cornea was lusterless and scaly and the parenchyma opaque. The infiltration was most marked in the pupillary area and in an area continuous with it, extending medially to the margin of the cornea between 1 and 3 o'clock and merging without any demarcation into the conjunctiva. The infiltrations over the other parts were of varying thickness.

The blood vessels from the conjunctiva entered the cornea from all sides, extending nearly to the center.

The bulbar conjunctiva laterally and medially was leathery and thick, thrown into folds and covered with a fatty material. The upper and the lower part were the healthier. The tarsal conjunctiva was velvety but marked by strands of fine fibrous tissue running irregularly.

The vision in this eye was counting of fingers at 10 inches (25 cm.). The lids were not thickened. Examination of conjunctival scrapings showed mostly epithelial debris, a few healthy cells and no bacteria except Bacillus xerosis.

Examination of the left eye showed xerosis and infiltration of the cornea more uniformly distributed but less marked, and there were fewer blood vessels. The conjunctiva presented a picture similar to that of the right eye, but the folds were not so marked.

On February 19 the application of sulfanilamide ointment twice a day in both eyes was begun.

On February 22 the right cornea had regained its luster in places and the scaliness had been replaced by foamy deposits scattered over the whole of the cornea. The infiltration had thinned out in places. The blood vessels were less conspicuous. The conjunctiva had regained some of its shining appearance; otherwise the condition was unchanged. In the left eye the normal appearance of the conjunctiva had been somewhat restored. The cornea was clearer and the dryness more apparent. The foamy deposits were less conspicuous than in the right eye.

On February 26 examination of the right cornea showed that from the periphery to about 4 mm. in the scaly appearance had disappeared and the infiltration had thinned out. In the central zone a strip about 2 mm. in breadth extending from 10 to 1 o'clock and another about 3 mm. in breadth from 5 to 8 o'clock were practically clear; the rest of the cornea was more opaque. There was also a general thinning of the opacity, and the luster had partially returned. Blood vessels were still present, but they were thinner. The conjunctiva was more supple, and the folds, though present, were less well defined. Of the left cornea, two strips, one triangular, with its vertex at 9 o'clock and its base, about 4 mm. long, crossing the center, and the other an oblong area about 2 mm. in breadth extending from the lateral margin of the pupillary area to the periphery and touching 1 o'clock and 2 o'clock, were denser than the rest. The vision was 2/60.

On March 5 the periphery of the right cornea had still further cleared. Elsewhere, except in the center, the uniformly hazy infiltration had resolved into small thin strips interspersed with more opaque dots of various sizes. In the central zone there was only a general thinning. The scaly appearance also had disappeared. The conjunctiva was almost normal, the scaly and greasy appearance not being very noticeable. Blood vessels were still present, but they were much thinner than before. The vision of the right eye was 3/60. There was a general thinning all over the left cornea, although denser dots were scattered throughout. The luster had completely returned. The conjunctiva was almost normal, and the vision in this eye was 6/12. The moisture had returned in both eyes.

On March 9 examination of the right eye revealed that at the periphery the uniform haziness was replaced by fine dots. The medial strip was absent, but the lateral triangular area and the central zone were still opaque, although the density had decreased. The thinning was especially noticeable at the lower quadrant of the pupillary area. The thickening of the conjunctiva had almost disappeared, and the folds were not well defined. The eye was moist. The vision in this eye was 4/60. In the left eye the deeper dots had thinned out and general haziness had decreased. The vision was 6/9.

The patient could not come to the clinic for four days. When he returned, the condition of the left eye was unchanged, but in the right eye there were signs that dryness was returning. He was given 5 drops of a vitamin A preparation twice a day. The ointment was again applied.

After a month's application of the drug, the right cornea did not improve further, but the former improvement was maintained. In the left eye there was further clearing of the dots in the periphery, but in the central portion the same condition was maintained. The patient was advised to have the treatment one week each month for three months. In the meantime, a contact glass was advised to prevent desiccation if possible.

LEUKOMA

Opacities of the cornea as a result of previous ulcerations are extremely common in India, since so many causes contribute to their formation. Unless the scar tissue is very dense and penetrates deeply into the posterior layers of the cornea, they are amenable to local application of sulfanilamide. Even opacities penetrating the whole depth of the cornea frequently thin out so that the vision improves.

CASE 10.—R. M., a Hindu woman aged 20, came to our clinic March 18, 1939, complaining of dimness of vision in the right eye. The left eye was completely blind. About nineteen years before she had had an attack of smallpox, which resulted in loss of the sight of both eyes. Subsequent prolonged treatment with medicines did not improve the vision, and at the age of 12 she had an operation done on the right eye. This slightly restored her vision.

Examination of the right cornea revealed slight ectasia of the central portion. There were three concentric zones of leukoma, the densest being a central pupillary zone about 6 mm. in diameter through which the pupil was invisible. Through the next zone, about 2 mm. in breadth, by strong focal illumination some idea of the iris could be obtained. The peripheral zone was clearer, and from 10 to 12 o'clock an artificial pupil could be indistinctly seen. Even in this area, which was the clearest portion of the cornea, there was a uniform haziness throughout, with smaller, denser areas lying both superficially and deeply in the corneal substance up to Descemet's membrane. The vision was counting fingers at 3 feet (91 cm.).

Examination of the left cornea revealed that it was opaque and slightly ectatic. The iris was not visible. No sign of any irritation was present. The vision was reduced to light perception and projection.

Treatment of the right eye only with sulfanilamide was instituted the day of admission. By March 20 some of the more superficial opacities at the periphery had disappeared. Vision was counting of fingers at 5 feet (152 cm.). On March 23 still further clearing of the superficial areas was seen. The anterior portion of the dots penetrating the posterior layers seemed to have disappeared. The dots situated in the posterior layers were unaffected. Vision was 5/36.

During the course of the next week the vision improved to 5/24, after which for a week there was no further improvement. Use of the sulfanilamide ointment was stopped at this stage, and 2 per cent ethylmorphine hydrochloride solution once a day and 0.5 per cent yellow mercuric oxide ointment twice a day were applied for three days. The patient could see the 5/24 line more distinctly now. Ethylmorphine hydrochloride and sulfanilamide ointment were used for about ten days, and the vision improved to 5/18. No further improvement was obtained even by application of the drugs for another fortnight. There were still scattered opacities deep in the cornea, the superficial areas having disappeared.

EXPERIMENTS

In view of the fact that sulfanilamide is effective in both the infective and the noninfective ocular conditions, it was apparent that besides its bacteriostatic effect it must have some action on the conjunctiva and on the cornea when applied locally. With this idea in mind we used it in the same strength (5 per cent) in the form of an ointment to the right eyes of 3 rabbits for four consecutive days morning and evening. On the morning of the fifth day it was once again applied, and the eyes were enucleated an hour later. The lids were also snipped off. The left eyes were used as controls. All the specimens were put into Bouin's fluid, and serial paraffin sections were made in the usual way. The four day period of application was chosen arbitrarily because if there was going to be any tissue reaction this period would be sufficient.

Clinically, within an hour the animals showed hyperemia of the palpebral conjunctiva and edema of the lids with redness of their margins. Practically, there was no congestion of the bulbar conjunctiva, nor could any change in the cornea be detected excepting a faint haziness. This condition persisted throughout the experiment. No effect on the iris or the pupil could be detected.

Histologic studies revealed the following changes, the reaction to the drug in the eyes of different animals of the same species being practically the same.

The Conjunctiva.—General hyperplasia of the epithelium was the rule, although in places normal epithelium was detected. The hyperplasia varied quantitatively. In some places it was many layered and flattened on the summit; in others it had a cauliflower-like appearance, being connected to the main body by a small stalk of epithelial cells; or it formed dome-shaped projections. Pillar formations and cup-shaped downward growths of epithelial cells were also seen. In the subepithelial layers there was a general infiltration with lymphocytes and endothelial cells, which in places formed dense masses. In some places they formed nodules. These infiltrations penetrated more or less up to the skin of the lids.

The blood vessels were dilated, and there was hyperactivity of the endothelial cells. Edema was especially well defined near the lid margins.

The Cornea.—Epithelium: There was a general tendency for the epithelium to be multiplied into many layers, especially near the central area, where the overgrowth led in some cases to a cockscomb-like appearance. At the periphery of the cornea the hyperplasia was at its minimum.

Bowman's Membrane: This did not show any appreciable change.

Substantia Propria: Here the most remarkable change was in the shape and the size of the corneal corpuscles. The cells were increased in size, and the protoplasm was rounded or irregular in shape. In no place was any cellular division noted. The hyperactivity was at its minimum near the periphery, where, however, there was a moderate amount of infiltration with lymphocytes. These changes were seen throughout the cornea except in the most posterior layers. About 90 per cent of the cells took part in the activity, although in places groups of normal cells could be detected.

Descemet's Membrane: This was unaffected.

Endothelium: There was no change.

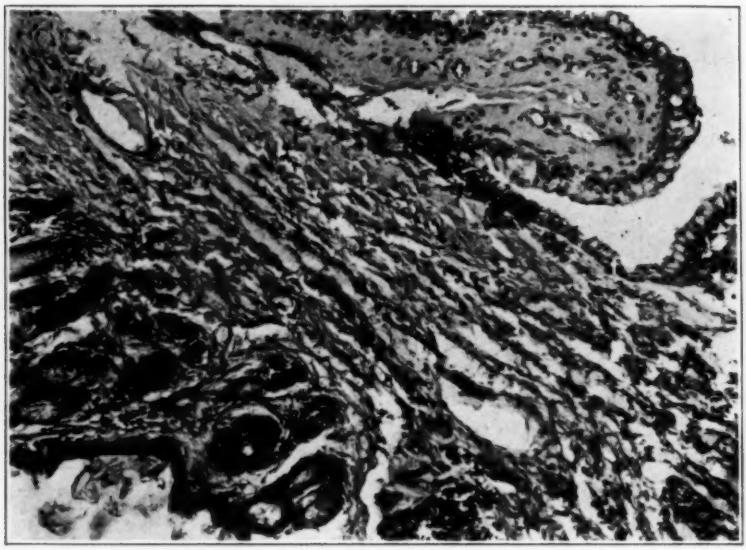

Fig. 9.—Normal conjunctiva of rabbit's eye.

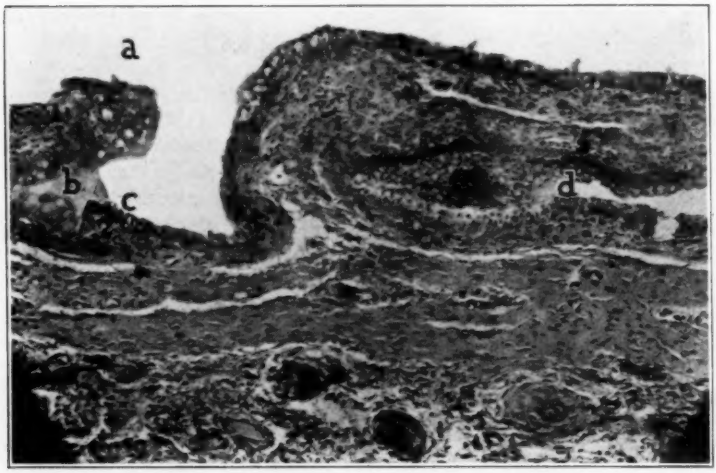

Fig. 10.—Rabbit's conjunctiva after application of sulfanilamide. Note (*a*) irregular hyperplasia (especially well defined at *a*, *b*, *c* and *d*), (*b*) increased infiltration throughout the conjunctiva, (*c*) dilation of the blood vessels and (*d*) proliferative activity of the endothelial cells.

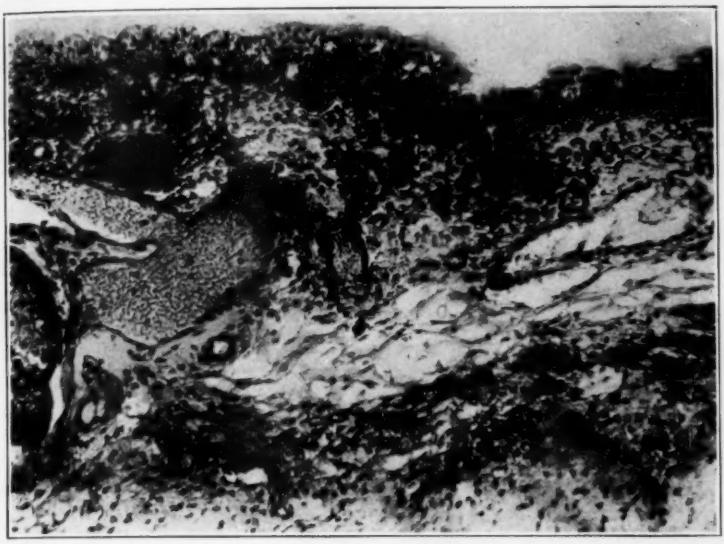

Fig. 11.—Rabbit's conjunctiva near the lid margin after application of sulfanilamide (iron-hematoxylin stain). Note dense infiltration throughout and edema.

Fig. 12.—Rabbit's conjunctiva after application of sulfanilamide, showing enormous hyperplasia of the epithelium.

EFFECT OF SULFANILAMIDE ON INCISED WOUNDS OF THE CORNEA

To determine the effect of the drug on incised wounds of the cornea the eyes of a rabbit were incised aseptically with a cataract knife in the center of the cornea. The incisions were about 5 mm. long. Prolapse

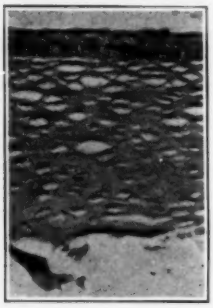

Fig. 13.—Normal cornea of rabbit's eye.

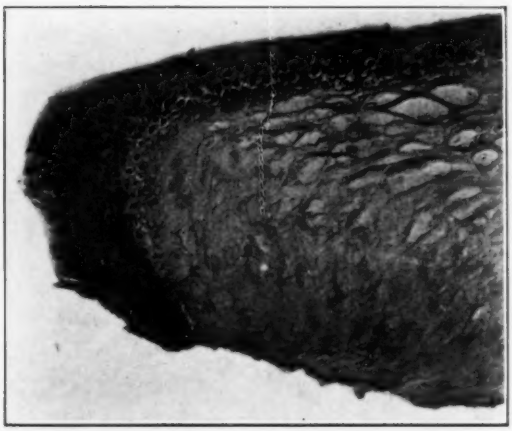

Fig. 14.—Rabbit's cornea after application of sulfanilamide (section near the center). Note hyperplasia of the epithelium and enlargement of the corneal corpuscles (the cornea was incurved during the process of embedding).

occurred in both eyes. Sulfanilamide ointment was applied to the right eye, the other eye being used as a control.

After twenty-four hours the right cornea appeared almost normal, with slight haziness. The incised wound was in apposition, and a white line on each side of it was visible under the corneal microscope. The rabbit could open this eye in a normal manner.

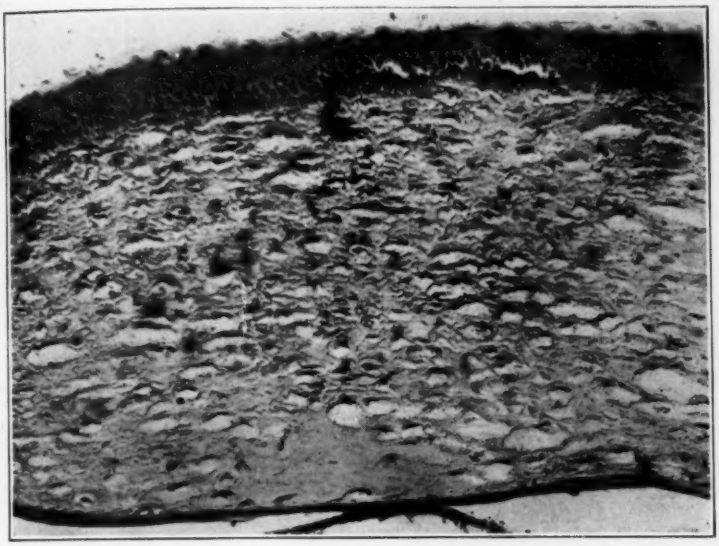

Fig. 15.—Rabbit's cornea after application of sulfanilamide (section near the middle third). Note moderate hyperplasia of the epithelium and change in shape of almost all the corpuscles.

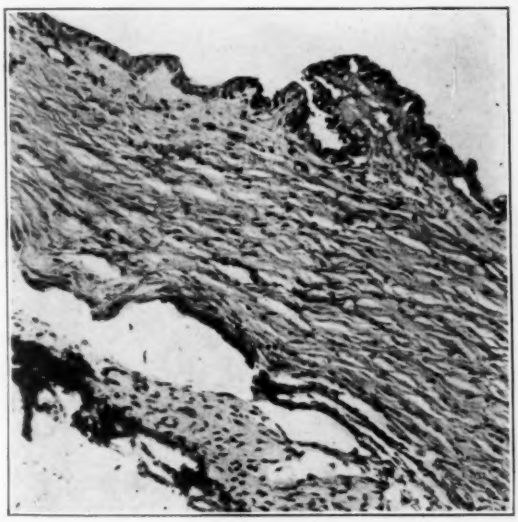

Fig. 16.—Normal cornea of rabbit's eye near the limbus.

Fig. 17.—Rabbit's cornea near the limbus after application of sulfanilamide. Note moderate infiltration and nearly normal epithelium.

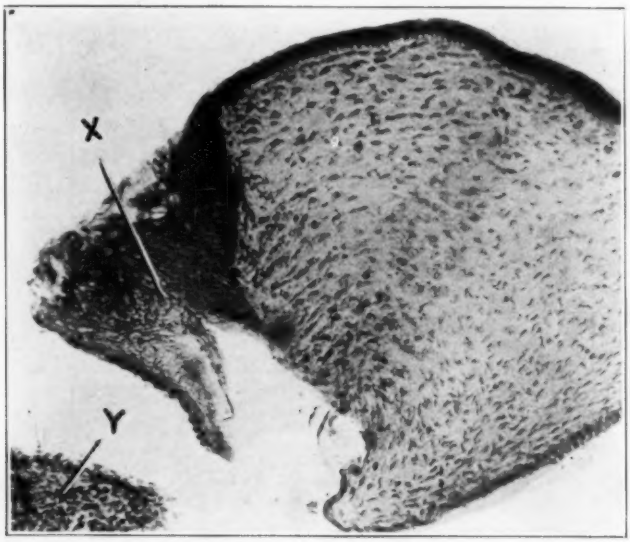

Fig. 18.—Normal incised cornea of rabbit's eye. Note that the coagulum (x) is incompletely replaced by epithelium. In the lower part there is no epithelium except at the margin. Most of the corpuscles are round. The iris (y) is densely infiltrated.

The left cornea was hazy throughout; the lips of the wound were swollen but in apposition. No white line was visible. The rabbit kept this eye almost closed.

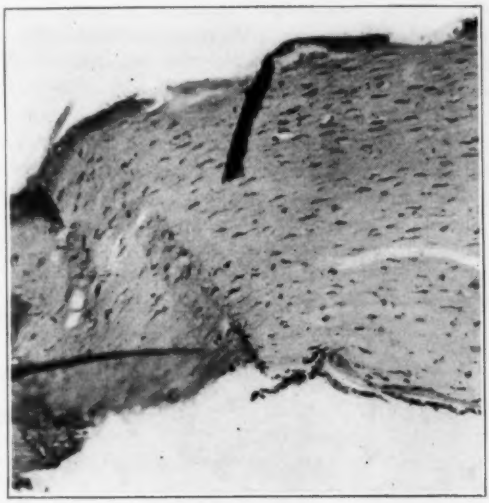

Fig. 19.—Incised cornea of rabbit's eye after application of sulfanilamide. Note that the coagulum has been almost completely replaced by epithelium anteriorly. Posteriorly the union is complete. The epithelium at the distal parts as well as the corpuscles is nearly normal.

Fig. 20.—Normal incised cornea of rabbit's eye near the limbus. Note the dense infiltration, especially at *a*, and the increased thickness of the epithelium.

Ciliary injection was less severe in the right eye than in the left eye. Histologic studies showed the following changes:

Eye Treated with Sulfanilamide.—Epithelium: Over the incision in the sections where the iris was not incarcerated and the cornea was apposed in the middle

portion, the coagulum was almost replaced by several layers of epithelium; in those in which the anterior portion was united complete bridging was noted. Posteriorly, the lips were generally separated and were seen in some sections still to be uncovered, while in the majority they were covered uniformly by a single layer of epithelium. In no section was the epithelium seen to pass beyond the cut margin posteriorly, although anteriorly heaping up above the surface was noted in some instances. In the sections in which the iris was caught between the lips of the wound, the surface anterior to it was filled with several layers of epithelium separated from the iris by a fine layer of coagulum, in which infiltration with epithelial cells could be noted. Immediately adjacent to the wound the epithelium was normal for a short distance on both sides; beyond there was slight irregular thickening up to the periphery. In some sections, on one side or the other of the incision normal epithelium was in evidence for the greater portion, while in others there was general hyperplasia of moderate intensity.

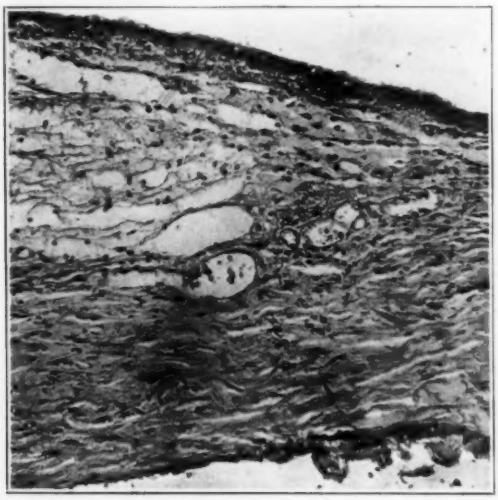

Fig. 21.—Incised cornea of rabbit's eye near the limbus after application of sulfanilamide. Note that the infiltration is much less than that in figure 20.

Substantia Propria: Infiltrations: Near the cut portion there was some infiltration with leukocytes and plasma cells which varied in different sections from scanty to moderate; the majority of the areas, however, showed few leukocytes. Sometimes it happened that on one side of the wound there was moderate infiltration while on the other few cells were seen. The leukocytes were few and far between in the rest of the cornea to the periphery.

Changes in corneal corpuscles: Near the incision the corneal substance showed a moderate number of round and oval or elongated forms of fibroblastic corpuscles. These varied in different sections. In some sections the majority of the corneal corpuscles were normal or showed slight thickening. In the sections where the union was complete, the corpuscles were almost normal in appearance, with few elongated or oval nuclei. The round forms in most of the sections, however, became scanty a short distance from the incision, so that by the midcentral zone only the normal and the elongated forms were visible. Beyond, almost all the

corpuscles had normal nuclei. In a few sections the hyperactivity proceeded to the midcentral zone.

Endothelium: This layer showed practically no reaction.

In the peripheral portion there was moderate infiltration with various types of cells from the iris. Anteriorly, the limbal vessels showed mild activity, and collections of endothelial cells and lymphocytes could be seen near these vessels.

The iris was incarcerated in some places, especially in the middle zone on one side, and no line of demarcation from the cornea could be detected.

Infiltration of the iris was slight.

Eye Not Treated with Sulfanilamide.—The scaffolding by the epithelium was not so complete, so that most of the coagulum remained uninvaded by epithelial cells; the majority of the sections showed hyperplasia of more uniform character and of greater intensity practically throughout the cornea about to the limbus.

Substantia Propria: The corneal substance showed a large number of rounded, oval and bizarre forms throughout the cornea nearly to the outer third. In the outer third the round forms were less in evidence, but other forms mixed with cells whose nuclei were only thickened could be seen. In some sections nearly normal corneal cells were present. There was a uniform infiltration with leukocytes without any particular localization near the incised portion.

Descemet's Membrane: This layer showed no change.

Endothelium: The endothelial cells showed hyperactivity in places.

Limbus: The limbus showed dense infiltration with cells from the iris, and this extended some distance to the cornea and a slight way toward the sclera. Evidently the repair work was just begun, the superficial bridging had been done and the deeper portion had been invaded; but the work was far from complete, so that it was not possible to check the defensive measures as yet.

COMMENT

A few common conditions of the eye were treated locally with sulfanilamide ointment; the results were interesting and at the same time encouraging.

The present method of treating trachoma is practically a failure, for reasons well known to every ophthalmic surgeon; the result is that many patients are condemned to a life of blindness. The effect of sulfanilamide on the granules is remarkable. They vanish with incredible rapidity. Especially interesting are the effects on the corneal complications of trachoma, for which there has been practically no treatment worth the name.

The treatment of keratoconjunctivitis with adenitis has presented equal difficulty. The resistant nature of the infective agent calls for strong remedies, but the generally acute course of the disease contraindicates such remedial measures as are used in the treatment of trachoma. The result is that the patient often remains half treated. It is true that the drugs generally used for acute conditions ameliorate the symptoms, but they fail in the majority of the cases to cure completely, with the consequence that a person who is once infected is always threatened with blindness.

Corneal complications, such as superficial or deep keratitis or ulcerations of the cornea, have always taxed the patience of both the surgeon and the patient, and the uncertainty of prognosis has been a dread to every practicing physician. The results obtained with sulfanilamide show that the ophthalmologist possesses in this remedy an agent which will at least enable him to do something for the patient and shorten the course of the disease. The most remarkable and important effect is the relief of symptoms, such as pain, photophobia, a gritty sensation and lacrimation, sometimes within a few hours of the first application of the drug and even before the clinical condition shows much improvement, and this without any irritation whatever.

Hitherto, dacryocystitis has been attributed mainly to disease of the paranasal sinuses or, in trachomatous countries, to trachoma. A consideration of the histologic changes induced in the sac by coccoid bodies when the condition is a complication of keratoconjunctivitis seems to indicate that the brunt of the toxin is borne by the inner segment of the sac, so that the best defined changes are in the epithelium, where hyperplasia, changes in the type of the cells and degeneration are conspicuous. The cellular infiltration and fibrosis resulting from chronic irritation are also confined to the inner portion. Therefore, when the pathologic process has not advanced too far and the obstruction is due mainly to edema and cellular infiltration, with a little fibroblastic reaction, the drug by removing the source of irritation is able in many cases to bring about a healthy condition of the sac.

The presence of eosinophilic cells in the cornea in cases of pannus associated with keratoconjunctivitis without any distinguishing sign of allergy is worthy of notice. The only other infective condition of the conjunctiva with which pannus is known to be associated is trachoma II-*b* (MacCallan [4]), and with this disease it is attributed to associated spring catarrh.

The failure of the drug to maintain its effect at the same level throughout its period of application is a handicap to a certain extent, but by the time it begins to lose its effectiveness the disease is already under control; thus the diminished action toward the end of the treatment is not of much practical importance.

The reason for this peculiar behavior is probably the fact that the drug enables nature to exert itself to its utmost within a short time but that the peak cannot be maintained for long. This theory is strengthened by the fact that an increase in the number of degenerated leukocytes together with the progressive decrease in the number of coccoid bodies in packed epithelial cells is seen in smear preparations for the first few days after application of the drug and that the leuko-

4. MacCallan, A. F.: Trachoma, London, Butterworth and Co., Ltd., 1936.

cytes then become fewer as well as healthier and the bacteria in each cell become fewer. After having attained this condition it takes a comparatively longer period to make the tissue bacteria free.

That the clinical observations are not likely to be wrong is also suggested by the effects of the drug on the normal conjunctiva and cornea of the rabbit as revealed by histologic studies. Both in the cornea and in the conjunctiva nature's method of defense is imitated: In the conjunctiva there are proliferation of the epithelium, infiltration of the subepithelial cells, dilatation of the blood vessels and edema, and in the cornea there are hyperplasia of the epithelium, change in the shape and the size of the corneal corpuscles and cellular infiltration at the periphery. Therefore, even if sulfanilamide had no bacteriostatic property it would ably defend the tissues in its role as a chemical excitant. With this role in mind, it is easy for one to understand the drug's effect in such conditions as xerosis and leukoma. In the cases of xerosis, reported, by stimulating new epithelial cells both in the cornea and in the conjunctiva it caused degenerated epithelial cells to be cast off and to appear as foamy deposits in the cornea; these being replaced by young healthy cells, the luster of the cornea and the conjunctiva was regained within a short time. The histiocytic reactions in the cornea and the peripheral cellular infiltration helped to restore the transparency of the cornea to a certain extent. In the conjunctiva the subepithelial cellular activity induced by sulfanilamide helped to free the ducts of some of the conjunctival and lacrimal glands and thus removed the leathery condition. The improvement was maintained in those cases in which the glands could be revived to at least partial functional activity. Such an improvement is a distinct gain in therapy.

In leukoma the cellular activity induced by the drug was responsible for the removal of the fibrous tissues, which acted as foreign bodies. Why the deeper opacities were not easily removed is not yet possible to explain; but, in view of the fact that for so long all the agents employed in treating leukoma were irritants and the results were obtained after a long, tedious course of treatment, frequently with recourse to operative methods, sulfanilamide is worth a trial.

It has been frequently observed that the peripheral opacities clear earlier than the central ones; probably the cellular infiltration at the limbus explains this.

The effect of the drug in postoperative sepsis is interesting. By exciting the local tissue cells still further the drug helps the usual cellular and humoral defensive mechanism and the infection is quickly brought under control, and once a healthy condition is brought about a reparative process is quickly induced. That the drug is capable of producing this effect even with a single application is supported by the results of histo-

logic studies of its effect on aseptic incised wounds of rabbits' corneas. The reports of Guyton, Butler and Harry support the clinical observations herein set forth.

Bailey and Saskin [5] have shown that sulfanilamide is valuable in the treatment of corneal ulcers other than those associated with trachoma or keratoconjunctivitis even when administered intravenously. However, in view of the experimental results, local application is probably more suitable, supplemented if necessary by intravenous administration in cases of severe ulceration.

It is not out of place to mention here that experiments are being carried out on the combined method of treatment—injections and local application—which seem to indicate that this method has more effect on the conjunctiva. On the cornea the effect seems to be not so pronounced.

The different preparations of sulfanilamide and its derivatives have somewhat varied effects on the cornea; on the conjunctiva their effects are nearly the same. A comparative statement will be published later.

86 Dhurrumtolla Street.

5. Bailey, J. H., and Saskin, E.: Treatment of Severe Corneal Ulcer with Sulfanilamide, Arch. Ophth. **22**:89-96 (July) 1939.

DETACHMENT OF THE PIGMENT BORDER OF THE IRIS

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The following case of detachment of the pigment border of the iris is of interest both because of the rarity of the lesion and because of the information related to other obscure conditions of the anterior segment that may be derived from it.

The patient, a 48 year old white man, a painter, was examined in July 1940 to determine whether blurred vision in the right eye resulted from a fall two months previously. The fall occurred when he lost his footing while working on a scaffold. He dropped 10 feet and suffered a skull fracture in the left occipital and parietal regions, with cerebral concussion, and an incomplete fracture of the right clavicle. He had had no ocular injuries and no previous ocular symptoms. He had never noted any structural ocular defects.

On ocular examination, a detachment of the pigment border of the iris was seen in the right eye, as shown in the accompanying illustration. The ropelike strands consisted of a series of nodular bodies which are called flocculi, granula, villositates, grape seed bodies or corpora nigra by various authors. They are enlargements of the normal pigment margin of the iris. The combined length of the two strands corresponded to the extent of the defect in the pigment border of the iris margin. The upper strand was in apposition with the anterior lens capsule and rubbed against it with each alteration in size of the pupil. There were three small clumps of iris pigment on the anterior capsule of the lens.

All other ocular findings were essentially normal. The cornea showed no pathologic changes, no punctate keratitis was present and the aqueous chamber was optically empty. Gonioscopy disclosed slightly increased pigmentation along the pigment line but no other abnormalities. The pupils were equal in size, reacted normally and dilated well with eucatropine hydrochloride. The iris sphincter and stroma were seen to be intact both on direct observation and on transillumination. No evidence of exfoliation of the anterior capsule of the lens could be seen. The media, fundus, visual field and blindspot were normal.

Refraction during eucatropine hydrochloride mydriasis disclosed the following error:

Right eye: $+1.50$ D. sph. $\frown +0.25$ D. cyl., axis 165
Left eye: $+0.50$ D. cyl., axis 105

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The muscle balance, ocular tension and accommodative power were normal.
The left eye was entirely normal.
The blurred vision in the right eye was due to the uncorrected refractive error.

It is of interest to consider the possible causes of this detachment of the pigment margin of the iris.

In view of the case history, trauma presents itself for first consideration. Yet there are several objections to accepting trauma as the etiologic agent.

Detachment of the pigment border of the iris.

The pigment border of the iris does not tear off in this manner even when posterior synechias are broken by powerful mydriatics.

The direct trauma incident to cataract extraction may rub off some of the pigment layer of the iris [1] but does not to my knowledge produce an actual detachment of the pigment border.

Indirect trauma may produce a tear of the sphincter muscle, an iridodialysis or dislocation of the lens and yet does not produce a detachment of the pigment margin.

1. Würdmann, H. V.: Injuries of the Eye, St. Louis, C. V. Mosby Company, 1932, p. 513.

In the only other similar case [2] which I have been able to find in the literature, the detachment was not associated with trauma or disease. The 38 year old patient had myopia of — 3.00 D., and ophthalmic examination gave negative results except to reveal changes at the iris margin. Slit lamp examination of the right eye disclosed a hypertrophied pigment border with round, irregular pigment excrescences, some of which were tumor-like. One excrescence was S shaped, extended downward over the anterior surface of the iris and wavered with movement. Several excrescences were also present on the pupillary border of the left eye, but, in addition, the pigment margin was detached over approximately a third of its circumference and hung downward from the upper margin of the pupil.

Although Dymschitz did not discuss the etiologic factor in his case, it probably was congenital.

In view of the foregoing remarks, it would seem probable that the cause of the detachment of the pupillary margin in my case was a congenital defect, even though trauma may have been the immediate precipitating factor.

It is well known that in the development of the iris the ectodermal portion is formed from the anterior and the posterior layer of the optic cup. As these layers begin to come into apposition, a narrow tubular channel remains at the periphery. This channel is known as the marginal sinus and is obviously the last portion of the optic vesicle to disappear.[3]

The marginal sinus increases in size as the iris grows, is largest at the fifth month of embryonic life and normally disappears by the seventh month. However, remnants occasionally persist into adult life. Congenital cysts of the iris contain cavities which communicate with the marginal sinus.[4] It is probable that the larger projections on the iris margin in Dymschitz' case communicated with the marginal sinus.

The marginal sinus is likewise persistent and relatively enlarged in those cases in which flocculi are present on the iris margin, as in the case presented. In such cases it expands anteriorly and is everted around the inner margin of the iris stroma. It is pure conjecture, but not unlikely, that the peripheral wall of the marginal sinus, which unites it

2. Dymschitz, L. A.: Ein Fall von ungewöhnlich stark entwickelten Flocculi iridum, Arch. f. Ophth. **127**:100, 1931.

3. Mann, I.: The Development of the Human Eye, London, Cambridge University Press, 1929, p. 119.

4. Mann, I.: Developmental Abnormalities of the Eye, London, Cambridge University Press, 1937, p. 171.

with the remainder of the iris ectoderm, would thus be weakened. The ropelike string of flocculi might then break away from the remainder of the iris ectoderm whenever the slightest trauma occurred.

This mechanism would also explain the origin of the chocolate-colored cysts which are occasionally seen floating freely in the anterior chamber.

It is interesting to note that the anterior capsule of the lens was entirely normal and that no evidence of shredding or flaking of the capsule had occurred despite the fact that an iris strand had rubbed on a given area of the capsule with each movement of the iris.

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PRIMARY ENDINGS OF THE OPTIC NERVE IN MAN AND IN ANIMALS

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It is of great value from time to time to compare recent advances in the study of human anatomy with such facts as were formerly known and accepted by scientists as definitely determined. The attempt is well justified in connection with the various terminations of the optic nerve, although many excellent papers on this subject, especially in regard to the primary endings of the nerve, have in the past been published: those on the experimental work of Minkowski,[1] Brouwer and Zeemann [2] and Putnam and Putnam [3]; those on the comparative studies of LeGros Clark,[4] Huber and Crosby [5] and Ariëns Kappers, Huber and Crosby,[6]

This investigation was aided by a grant from the Rockefeller Foundation.

From the Department of Neurology, Columbia University College of Physicians and Surgeons.

1. Minkowski, M.: Ueber den Verlauf, die Endigung und die centrale Repraesentation von gekreuzten und ungekreuzten Sehnervenfasern bei einigen Säugetieren und beim Menschen, Schweiz. Arch. f. Neurol. u. Psychiat. **6**:201, 1920; **7**:298, 1920.

2. Brouwer, B., and Zeemann, W. P. C.: The Projection of the Retina in Primary Optic Neuron in Monkeys, Brain **49**:1, 1926; Experimentell-anatomische Untersuchungen ueber die Projektion der Retina auf die primären Opticuscentren, Schweiz. Arch. f. Neurol. u. Psychiat. **13**:118, 1923.

3. (*a*) Putnam, T. J.: Studies on the Central Visual System: II. Comparative Study of Form of Geniculostriate Visual System of Mammals, Arch. Neurol. & Psychiat. **16**:285 (Sept.) 1926; (*b*) IV. Details of Organization of Geniculostriate System in Man, ibid. **16**:683 (Dec.) 1926; (*c*) Studies on the Central Visual Connections, ibid. **16**:566 (Nov.) 1926. (*d*) Putnam, T. J., and Putnam, I. K.: Studies on the Central Visual System: I. Anatomic Projection of Retinal Quadrants on Striate Cortex of Rabbit, ibid. **16**:1 (July) 1926.

4. Clark, W. E. L.: (*a*) The Structure and Connections of the Thalamus, Brain **55**:406, 1932; (*b*) An Experimental Study of Thalamic Connections in the Rat, Phil. Tr. Roy. Soc., London, s.B **222**:1, 1932; (*c*) A Morphological Study of the Lateral Geniculate Body, Brit. J. Physiol. Optics, May 1932; cited by Ariëns Kappers, Huber and Crosby [6]; (*d*) The Medial Geniculate Body and the Ganglion Isthmi, J. Anat. **67**:536, 1933.

5. Huber, G. K., and Crosby, E. C.: The Nuclei and Fibre Paths of the Avian Diencephalon with Consideration of Telencephalic and Certain Mesencephalic Centers and Connections, J. Comp. Neurol. **48**:1, 1929; Somatic and Visceral Connections of the Diencephalon, Arch. Neurol. & Psychiat. **22**:187 (Aug.) 1929.

6. Ariëns Kappers, C. U.; Huber, G. K., and Crosby, E. C.: The Comparative Anatomy of the Nervous System of Vertebrates Including Man, New York, The Macmillan Company, 1936, vol. 2.

and others which will be quoted hereafter. Thorough studies which I have made have revealed several new facts discussed in this paper.

It may first be predicated that all the fibers of the optic nerve arise in the retina; the existence of antidromic fibers, as suggested by previous investigators, e. g. Perlia,[7] Wallenberg[8] and Jelgersma,[9] has been denied by Muenzer and Wiener,[10] and I agree with the conclusions reached by these authors as a result of study of the complete destruction of one anterior colliculus in a rabbit in which no sign of degeneration in the optic tracts, chiasm or nerves could be found.

The most important primary end station of the optic nerve is the lateral geniculate body. There is no doubt that in man almost 80 per cent of the optic fibers enter this mass of gray matter. The number of optic fibers which terminate in the lateral geniculate body is in direct proportion to the position of the animal in the phyletic scale; the more highly developed the animal, the greater the number of fibers ending in this gray mass. The subdivision of the lateral geniculate body into a dorsal and a ventral ganglion is true of all animals (the Putnams,[3] S. Környey[11]), the ventral ganglion being transformed into the griseum praegeniculatum (Minkowski[1]) or, in the monkey and in man, the griseum parageniculatum (Balado and Franke[12]). While the dorsal ganglion represents the cortical relation, the ventral is connected only with the anterior colliculus (the Putnams[3]).

As for the second nucleus, the pulvinar, there is doubt whether it is a primary end station for retinal fibers; my comparative anatomic studies facilitate the decision. A homologization of the pulvinar in man with a similar nuclear mass in birds seems difficult. The nucleus dorsalis thalami lateralis in birds (Huber and Crosby[5]) may in its posterior part be a forerunner of the pulvinar, as may the nucleus lateralis thalami pars posterior in lower-developed mammals. But there is the nucleus

7. Perlia: Ueber ein neues Opticuscentrum beim Huhn, Arch. f. Ophth. (pt. 1) **35**:20, 1889.

8. Wallenberg, A.: Das mediale Opticusbündel der Taube, Neurol. Centralbl. **17**:532, 1898.

9. Jelgersma, G.: Die sensiblen und sensorischen Nervenbahnen und Centren, Neurol. Centralbl. **14**:290, 1895.

10. Muenzer, E., and Wiener, H.: Das Zwischen- und Mittelhirn des Kaninchens und die Beziehungen dieser Teile zum übrigen Centralnervensystem, Monatschr. f. Psychiat. u. Neurol. **12**:241, 1902.

11. Környey, S.: Zur vergleichenden Morphologie des lateralen Kniehöckers der Säugetiere, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **30**:93, 1927.

12. Balado, M., and Franke, E.: Das Corpus geniculatum externum. Eine anatomisch-klinische Studie, Monographien aus dem Gesamtgebiete der Neurologie und Psychiatrie, Berlin, Julius Springer, 1937, p. 62.

posterior of Koelliker (Nissl's[13] nucleus posterolateralis; Ramon y Cajal's[14] "noyau thalamique postérieur ou foyer prébigéminal"), medial to the nucleus lateralis pars posterior at the same level. Into this nucleus of Koelliker enter retinal fibers, or fibers originating in the lateral geniculate body. I found this nucleus posterior, well developed in rodents, in the kangaroo and in the tapir to be separated from the pulvinar by a shallow groove. In man as well as in highly organized animals the posterior nucleus has merged in the pulvinar. Thus a nucleus posterior proper as observed in the carnivores cannot be recognized in man and in the monkey. The nucleus limitans of the pulvinar, however, bordering the pulvinar medially, consists of larger cells than the pulvinar itself and resembles the nucleus posterior as to the size and shape of these cells. When it is considered that this nucleus posterior is a primary end station for optic fibers and that its coalescence with the pulvinar is, according to LeGros Clark,[15] an obvious fact, then one has to agree that the pulvinar also receives direct fibers from the retina.

There are three different opinions concerning this question: Minkowski[1] assumed a primary ending of optic fibers in the pulvinar; Brouwer and Zeemann,[2] especially the former, on the grounds of exact investigations in the monkey, denied this and stated the belief that the optic fibers pass by the pulvinar, whereas Glorieux[16] and Lorente de Nó[17] expressed the opinion that unmyelinated optic fibers enter and end in the pulvinar. In view of the fact that my Bielschowsky preparations of this region show that fibers from the brachium of the anterior colliculus bend upward, it is not likely that they are merely passing instead of entering the pulvinar. But it is still undetermined whether these fibers originate from the lateral geniculate body or from the retina (nucleus magnocellularis tractus optici, to be discussed hereafter). Anyhow, it seems that the question of whether optic fibers end in the pulvinar can be answered in the affirmative.

The optic thalamus is supposed to receive another contingent of retinal fibers, the fasciculus accessorius opticus anterior, whereas the fibers running in the basal optic root caudad to the ectomammillary

13. Nissl, F.: Die Kerne des Thalamus beim Kaninchen, Verhandl. d. Gesellsch. deutsch. Naturf. u. Aerzte (1889) **62**:509, 1890; Die Grosshirnanteile des Kaninchens, Arch. f. Psychiat. **52**:867, 1913.

14. Ramón y Cajal, S.: Histologie du système nerveux de l'homme et des vertèbres, Paris, A. Maloine, 1911, vol. 2.

15. Clark, W. E. L. (footnotes 4 *a* and *b*).

16. Glorieux, P.: Anatomie et connexions thalamiques chez le chien, J. de neurol. **29**:525, 1929.

17. Lorente de Nó, cited by Ariëns Kappers, Huber and Crosby.[6]

ganglion form the fasciculus accessorius opticus posterior (Bochenek [18]). The reception nucleus of the former group is said to be the lateral part of the corpus subthalamicum. Pavlov [19] and later Loepp [20] shared this opinion. Kosaka and Hiraiwa,[21] on the contrary, stated the belief that there are perhaps aberrant optic fibers which pursue their way around the peduncle, as described by Bochenek,[18] returning, however, laterally to the optic tract without entering into the corpus subthalamicum. LeGros Clark [15] could not prove such an ending with certainty; he wrote, "It is a differentiated lateral segment of the subthalamus and is connected with the zona incerta." It seems that he agreed with Ramón y Cajal,[14] who described, under the name "noyau de la bandelette optique," a nucleus medial to the optic tract which bulges the peduncle inward; this nucleus is seen in the mouse and in the rabbit at the level of and in close relation to the corpus subthalamicum. I have seen it in other animals. Spiegel and Zweig [22] suggested that it may be one of the lateral nuclei of the tuber cinereum. The nucleus of the ansa, described by me [23] may also belong in this group. In any case, the nucleus described by Ramón y Cajal [14] belongs to the hypothalamus, not the subthalamus. In their paper on the hypothalamus, Roussy and Mosinger [24] suggested retino-subthalamic fibers arising from the homolateral and the contralateral retina.

On careful examination of the optic tract in different mammals, including man, in dark-stained Weigert preparations, fibers of the finest caliber may be seen to arise from the interior of the tract. They enter into the spaces between the fascicles of the peduncle (fig. 1). The ending of these fibers is lateral in the subthalamic body. The lateral part of the

18. Bochenek, A.: Ueber zentrale Endigungen des Nervus opticus, Bull. internat. Acad. d. sc. de Cracovie (1908), 1909, p. 91.

19. Pavlov, M.: Les connexions centrales du nerf optique chez le lapin, Névraxe **1**:235, 1900.

20. Loepp, W. H.: Ueber die zentralen Opticusendigungen beim Kaninchen, Anat. Anz. **40**:309, 1912.

21. Kosaka, K., and Hiraiwa, K.: Zur Anatomie der Sehnervenbahnen und ihrer Zentren, Folia neurobiol. **9**:367, 1915.

22. Spiegel, E. A., and Zweig, H.: Zur Cytoarchitektonik des Tuber cinereum, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **22**:278, 1917.

23. Marburg, O.: Vergleichend anatomische Studien über den Nucleus hypothalamicus und die hypothalamische Striatumfaserung, Jahrb. f. Psychiat. u. Neurol. **38**:184, 1917.

24. Roussy, G., and Mosinger, M.: Étude anatomique et physiologique de l'hypothalamus, Rev. neurol. **1**:848, 1934; Sur le noyau tangentiel de l'hypothalamus et ses connexions, ibid. **2**:651, 1934; La systémisation du système nerveux et les correlations entre le système neuro-somatique et le système neuro-végétatif, J. de physiol. et de path. gén. **34**:486, 1936.

subthalamic body stains clearer than the more medial parts. This observation confirms all the suggestions of a direct retinosubthalamic fascicle.

As is probably true of the thalamus, and obviously of the subthalamus, the hypothalamus receives optic fibers. Edinger [25] found that after enucleation of the eye in a squirrel degenerated fibers entered into a nucleus which is now recognized to be the nucleus supraopticus. Roussy and Mosinger [24] and also Laruelle [26] assumed that at least collaterals from the optic nerve end in the nucleus supraopticus. This theory does not agree with the findings of Greving,[27] who suggested that these optic fibers leaving the anterior border of the chiasm pass through the nucleus

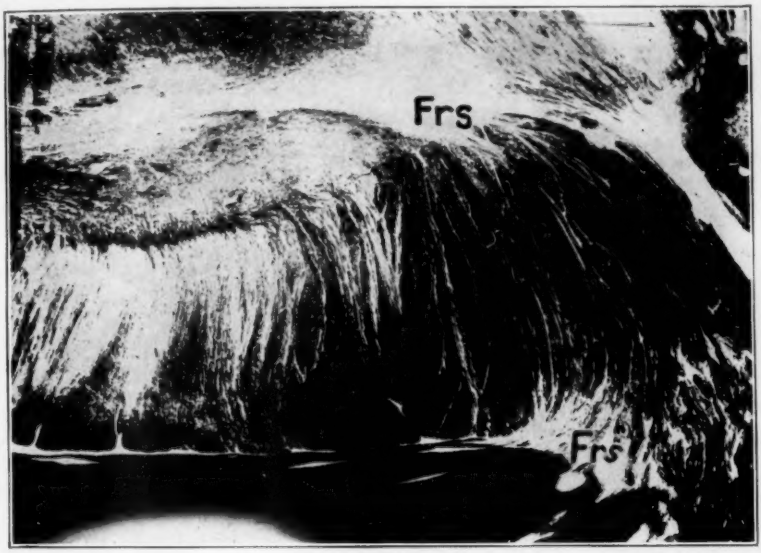

Fig. 1.—Fasciculus retinosubthalamicus (*Frs*); ventral to the peduncle, the optic tract; dorsally, the subthalamic body.

supraopticus without ending there, despite their branching in the nucleus, and end in the optic thalamus (the medial and the anterior nucleus).

I have seen these fibers in sagittal sections of the brain stem, where they can be followed either into the supraoptic nucleus or, passing this nucleus, into the optic thalamus. The second tract described by

25. Edinger, L.: Vorlesungen über den Bau der nervösen Zentralorgane, ed. 7, Leipzig, F. C. W. Vogel, 1908, vol. 1; ed. 8, 1911, vol. 2.

26. Laruelle, L.: Le système végétatif méso-diencéphalique, partie anatomique, Rev. neurol. **1**:809, 1934.

27. Greving, R.: Die zentralen Anteile des vegetativen Nervensystems, in von Möllendorf, W.: Handbuch der mikroskopischen Anatomie des Menschen, Berlin, Julius Springer, 1928, vol. 4.

Greving [27]—tractus opticothalamicus—arises from the chiasm posteriorly and ends also in the optic thalamus. These two, or better said three, tracts should more precisely be named tractus retinosupraopticus and tractus retinothalamicus, anterior and posterior. Only the first mentioned tract is hypothalamic. But there are two more hypothalamic connections. One, described by Frey,[28] leaves the optic tract behind the chiasm, forms a slightly curved arc to the medial side and enters into the medial tuber nuclei. I was in a position to confirm this tract in man

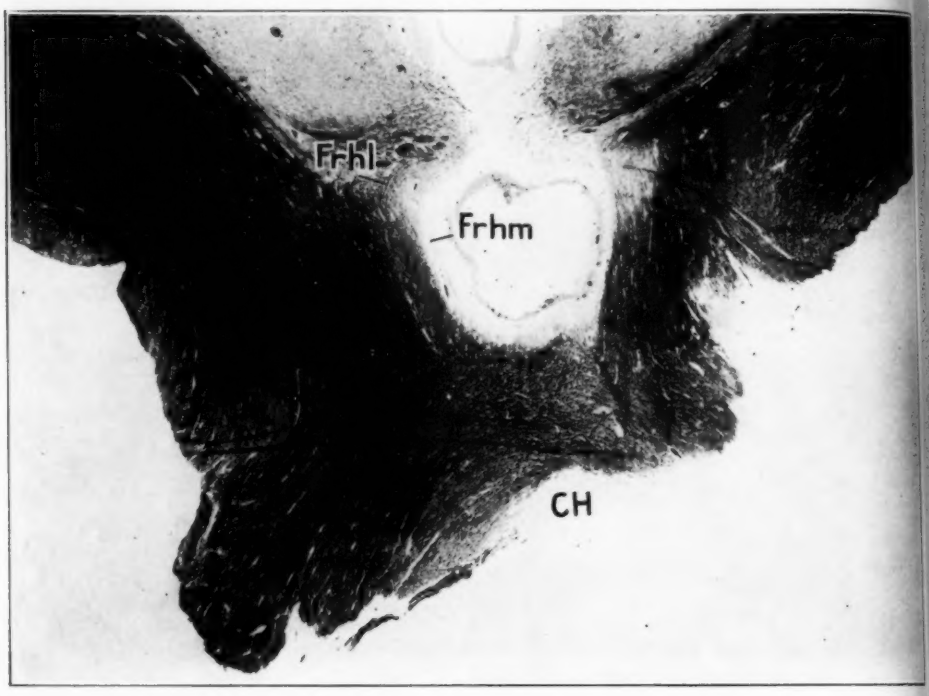

Fig. 2.—*CH,* horizontal section of the chiasm in man; *Frhl,* fasciculus retinohypothalamicus lateralis; *Frhm,* fasciculus retinohypothalamicus medialis.

(fig. 2). A second tract observed by me leaves the chiasm in its whole length and then forms a commissure-like bundle. The end of this bundle spreads in the lateral tuber nuclei, where it forms a fine network, thus indicating the termination. The bundle is well pronounced in birds (emu, sea gull; figs. 3, 4 and 5) and less well pronounced in mammals, including man. It is not absolutely certain whether these fibers seen in the emu are fibers of retinal origin, but their site, their

28. Frey, E.: Vergleichend anatomische Untersuchungen über die basale optische Wurzel, Zurich, Orell Fuessli, 1937.

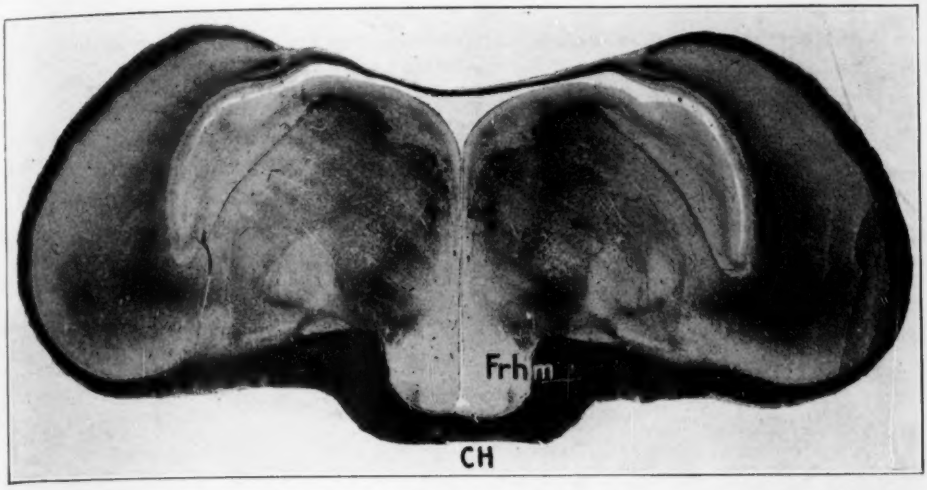

Fig. 3.—Region of the chiasm in the emu; above the chiasm (*CH*), a supraoptic decussation consisting of the finest fibers, which enter into a nucleus of the same site as the supraoptic nucleus (*Frhm*).

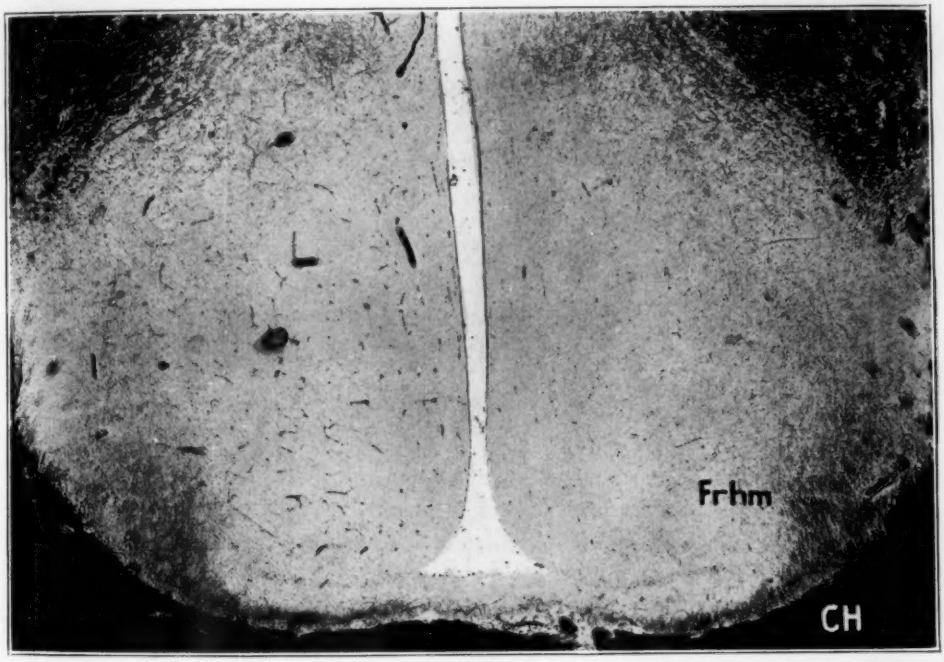

Fig. 4.—Detail from figure 3, showing the fibers emerging from the chiasm.

emerging from the interior of the optic nerve and their difference from the other well known supraoptic commissures are so definite that they may be assumed to represent a particular pathway not described hitherto. Thus there are three hypothalamic connections: the retinosupraoptic tract, the medial retinotuberal tract of Frey [28] (called hypothalamic optic root) and a lateral retinotuberal tract which I found in birds and also in

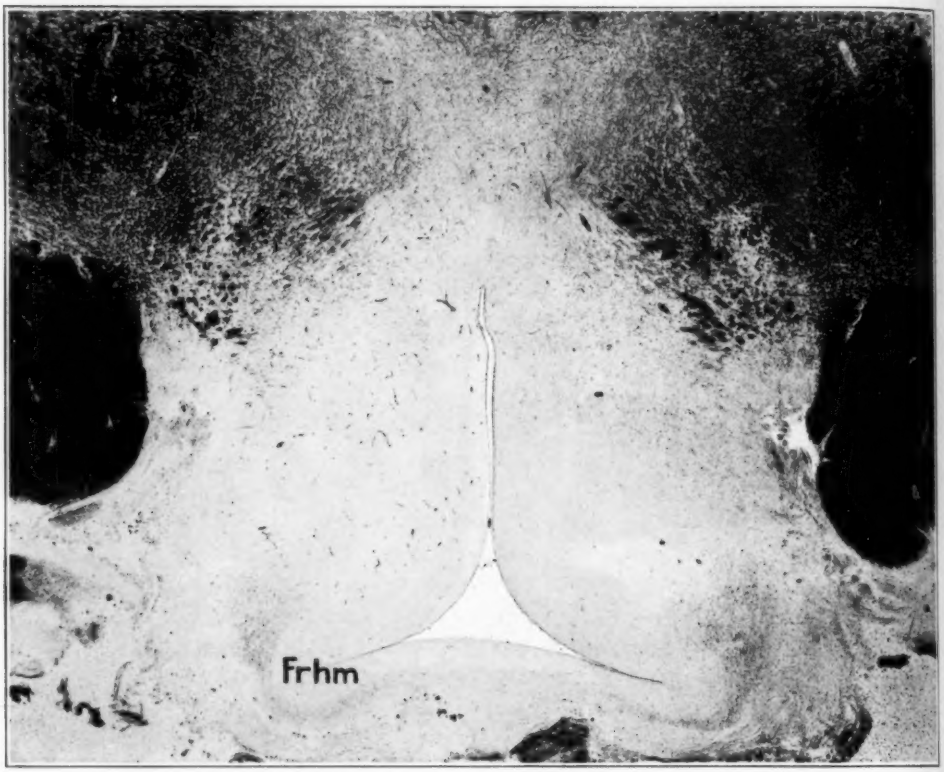

Fig. 5.—Behind the chiasm in the emu (ventrally a few fibers of it can be seen) the decussation forms a bundle lateral to the hypothalamus, *Frhm*. The optic tract lies beside the tuber; medial to it are the transverse-cut bundles of the tractus infundibuli.

mammals, the connection of which to the retina is, however, not yet proved.

In addition, there is a direct retinohypophysial tract which I studied together with Martha Geiringer.[29] This is a direct bundle emerging from the chiasm near the midline and bending ventrally to reach the

29. Geiringer, M.: Die Beziehungen der basalen Optikuswurzel zur Hypophyse und ihre Bedeutung für den Farbwechsel der Amphibien, Anat. Anz. **86**:177, 1938.

stalk of the hypophysis. I observed this tract in that part of the stalk in which Bochenek [18] described the hypothalamic fibers ending in the posterior part (glandula infundibularis) of the hypophysis. Since the destruction of the stalk in the frog results in a loss of color change depending on optic impressions, one must assume that the retinohypophysial tract is the afferent limb of a reflex arc serving this purpose. This direct connection is interrupted in mammals, including man, by the hypothalamic nuclei, thus forming a supraopticohypophysial tract and a tuberohypophysial tract.

From these descriptions it is to be recognized that the optic nerve appears to be connected with many parts of the diencephalon. The thalamus itself receives, according to Greving,[27] direct retinal fibers, and according to my view so does the pulvinar by means of retinothalamic fibers.

In addition there is evidence of the existence of a fasciculus retinosubthalamicus, which, although still under discussion, appears to be probable. As a third connection, the fasciculi retinohypothalamici (fasciculus retinosupraopticus, fasciculus retinotuberalis medialis and lateralis) are to be mentioned, with their continuations into the posterior hypophysial lobe, whereas in Amphibia there is a direct retinohypophysial connection. The entire metathalamus with the lateral geniculate body, consisting of a dorsal and a ventral part, receives the fasciculus retinogeniculatus, which increases in proportion with the development of the forms in an ascending scale; this special visual nucleus, the dorsal nucleus, transfers impressions from the optic nerve to the calcarine neocortex, which is already present in birds (fig. 6).

While the lateral geniculate body in its dorsal ganglion appears to increase in complexity and size in an ascending scale, the contrary is the case with the superior colliculus. In this mass of gray matter in all vertebrates, including man, may be distinguished (1) the colliculus itself, the tectum; (2) the transitional part from the optic thalamus to the colliculus, the pretectum; (3) the so-called ganglion isthmi, with its optico-acoustic relations; (4) Fuse's "gewundenes Grau," or "colliculular olive," lateral in the tectal area near the brachium of the anterior colliculus, and (5) outside of the tectal region, the ectomammillary ganglion of Edinger, with its varying nomenclature. The stratum opticum of the tectum consists of retinal and occipital fibers, the former ending in the griseum primum (superficiale). As stated by Berl [30] and the Putnams,[3d] the stratum zonale is in connection neither with the retina nor with the occipital lobe.

30. Berl, V.: Einiges über die Beziehungen der Sehbahnen zum vorderen Vierhügel der Kaninchen, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **8**:308, 1902.

Concerning the ganglion isthmi, there is the medial optic root, or central optic bundle, which connects this ganglion with the retina (Perlia,[7] Wallenberg,[8] Jelgersma[9]). Within this medial bundle, according to some writers there are antidromic fibers, the tractus isthmo-opticus (a term also used by Craigie[31] and Huber and Crosby[5]); these fibers are supposed to impede or instigate a retinal irritation by light (Wallenberg[8]). One has to agree with Muenzer and Wiener[10] that such an antidromic connection does not exist. The ganglion isthmi consists, as is well known (LeGros Clark[4d]), of two parts, a parvicellular part connected with the cochlear nerve and a magnocellular part in connection with the optic nerve via the medial optic root. Whereas the parvicellular

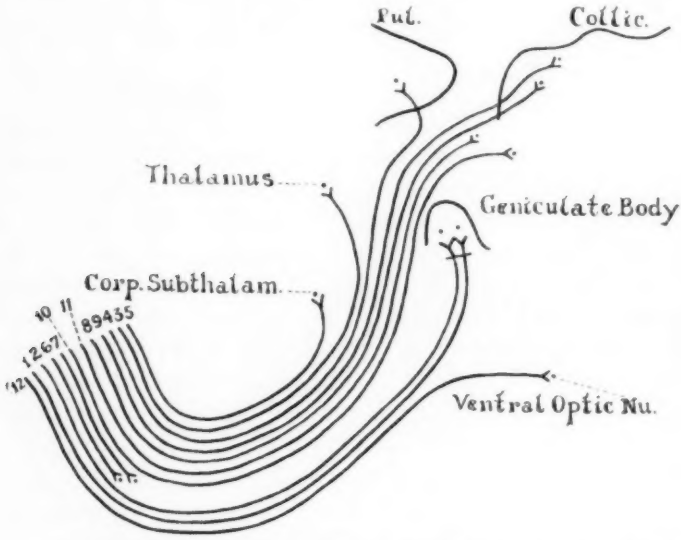

Fig. 6.—*1*, fasciculus retinogeniculatus; *2*, fasciculus retinopraegeniculatus; *3*, fasciculus retinothalamicus; *4*, fasciculus retinopulvinaris; *5*, fasciculus retinosubthalamicus; *6*, fasciculus retinosupraopticus; *7*, fasciculus retinotuberis (hypothalamicus medialis and lateralis); *8*, fasciculus retinotectalis; *9*, fasciculus retinoisthmicus (in man?); *10*, fasciculus from the retina to the collicular olive of Fuse; *11*, fasciculus retinopraetectalis to the dorsolateral optic nucleus of the midbrain, and *12*, fasciculus retino-opticoventralis (basal optic root).

part of this ganglion in higher animals is homologized by some writers with the nucleus dorsalis of the lateral lemniscus, the magnocellular part had not yet been observed in higher animals. Thus I started from the fact that a medial optic root can be demonstrated, as in birds, so also in mammals, even including man (fig. 7). This root begins with a few

31. Craigie, F. H.: The Cell Masses in the Diencephalon of the Humming Bird, Verhandl. d. k. Akad. v. Wetensch. **34**:1038, 1931.

fibers in the pretectal area and ends near the middle of the tectum opticum. The fibers of the brachium of the superior colliculus pass this colliculus as far as its medial border and turn here in a sagittal direction to form this small bundle. Thus in coronal sections one sees transversely cut fibers almost as far as the middle of the colliculus. But these fibers do not remain in this position: They turn ventrolaterally to disappear near the mesial end of the superficial gray matter.

When these regions are examined in Nissl-stained preparations, large ganglion cells can be seen not only in the deep gray matter but

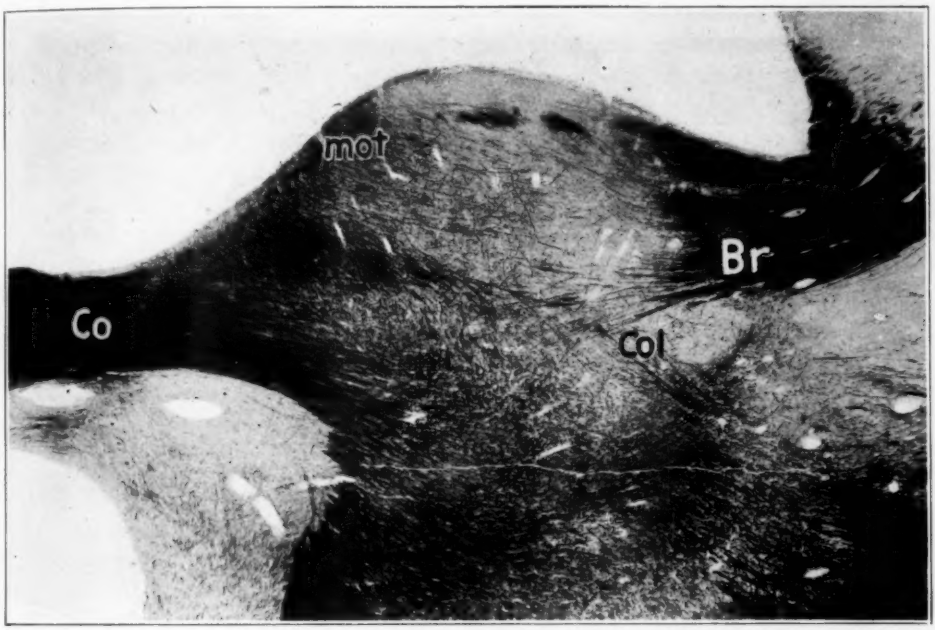

Fig. 7.—The anterior colliculus in man. Lateral, the brachium (*Br*) of this colliculus; medial, the posterior commissure (*Co*); running medially, forming a small medial (central) optic tract bundle, fibers of the brachium (*mot*); ventral from the brachium at the level of the posterior commissure, a clear spot, the collicular olive of Fuse (*Col.*)

in the more superficial layers.[32] It is not unlikely that they represent the long-sought magnocellular nucleus of the ganglion isthmi in mammals. Even if this medial bundle of the brachium in mammals represents the medial optic root of birds, there is no evidence that these fibers originate in the retina and not in the lateral geniculate body. Only the extreme resemblance in site, amount, shape and course of the fibers

32. Marburg, O.: Mikroskopisch-topographischer Atlas des menschlichen Zentralnervensystems, ed. 3, Leipzig, Franz Deuticke, 1927, fig. 17.

justifies the assumption of a structure equivalent to the medial root of the optic nerve.

The term pretectum is not to be taken in a too literal sense. There are nuclei which lie more on the thalamic side of the pretectal region and other nuclei which are anatomically more closely identified with the tectum. The differences in the development of the tectum, the thalamus and especially the pulvinar in more highly developed forms permit the recognition of an area which deserves the adjective pretectal. To find the connections of the optic nerve with the pretectal region in man, I first investigated this area in birds. Of the many nuclei described here, only two have to be taken into consideration: the spiriform nucleus, in direct connection with the optic nerve, and the pretectal nucleus, in connection with the lateral geniculate body. In birds these nuclei are found rather lateral and ventral to the tegmentum, because of the more lateral position of the tectum as compared with the tegmentum, whereas in higher-developed animals the tectum lies dorsally. Thus the nucleus spiriformis or its equivalent may appear to be shifted dorsally and situated lateral to a similarly dorsally and medially shifted pretectal nucleus, which presents in reality a pretectal area rather than a single nucleus.

In view of these features, two nuclei in the more highly developed forms may be identified: 1. A lateral nucleus, the magnocellular nucleus of the optic tract (LeGros Clark [33]), which is pretectal in position, ventral to the angle between the pulvinar and the tectum, and often characterized by the presence of an overlying blood vessel. This nucleus can be found in the vicinity of the habenular nucleus and receives optic fibers. It is not unlikely that it can be homologized with the nucleus spiriformis or a part of this nucleus.

2. The pretectal nucleus (LeGros Clark [33]), which can be seen at the level of disappearance of the habenulointerpeduncular tract, corresponds to the upper termination of this tract and is homologous with the respective nucleus in the lower-developed animals.

A discussion of these nuclei can be understood only when it is realized that the pretectal area does not present distinct nuclei in all forms but often presents only diffuse scattered cells with local accumulations of the cellular elements. Occasionally the nuclei are more definitely developed, for instance in the armadillo, as described by Papez,[34] who found, in addition to the magnocellular nucleus of the optic tract and a pretectal nucleus surrounded by rather scattered cells (area praetectalis),

33. Clark, W. E. L. (footnotes 4 *a*, *b* and *c*).

34. Papez, J. W.: (*a*) The Thalamic Nuclei of the Nine-Banded Armadillo (Tatusia Novemcincta), J. Comp. Neurol. **56**:49, 1932; (*b*) Connections of the Pulvinar, Arch. Neurol. & Psychiat. **41**:277 (Feb.) 1939; (*c*) Comparative Neurology, New York, T. Y. Crowell Company, 1929.

and situated ventral to these nuclei, a posterior nucleus. The same situation was described more or less definitely in papers by Rioch,[35] Gurdjian,[36] Tsai,[37] Chu [38] and Ingram, Hannet and Ranson.[39] Unfortunately, the nomenclature for the various nuclei is not identical in these papers, so that it is often difficult to homologize the different cell groups. In the Winkler-Potter atlas of the cat's brain,[40] the same situation may be seen to exist: Medial to the pulvinar there is the large posterior nucleus, while medial to this posterior nucleus and lateral to the habenular nuclei, two nuclei can be recognized. There is no doubt that these two nuclei can be homologized with the magnocellular nucleus of the optic tract attached to the nucleus posterior and with the pretectal nucleus and the pretectal area of Papez,[34a] lying nearer to the habenular nuclei and more ventromedially.

In order to decide this question as it relates to man, a series of sections was cut from the caudal portion of the optic thalamus as far caudally as the middle of the superior colliculus. The same situation as that described by Papez [34a] in the armadillo was found in man. In the region of the caudal portion of the habenular nucleus, in the angle between the pulvinar and the pretectal region, a small nucleus may be seen, dorsally covered by a blood vessel (fig. 8). This small group of cells could be followed through ten to twelve sections cut 20 microns thick. It is accompanied in its caudad portion by scattered cells of the real pretectal area (fig. 9). Ventrolaterally, the cells are a little larger and form a group with an indefinite border. Thus the conditions in man are the same as in the armadillo. The magnocellular nucleus of the optic tract is accompanied ventrally by smaller cells merging into the pulvinar. The connections of these two nuclei in man remain still to be proved, and they will be difficult of proof in view of the small number of cells.

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35. Rioch, D. M.: Studies on the Diencephalon of Carnivora: I. Nuclear Configuration of Thalamus, Epithalamus and Hypothalamus of Dog and Cat, J. Comp. Neurol. **49**:1, 1929.

36. Gurdjian, E. S.: The Diencephalon of the Albino Rat: Studies on Brain of Rat, J. Comp. Neurol. **43**:1, 1927.

37. Tsai, C.: The Optic Tracts and Centers of the Opossum, Didelphis Virginiana, J. Comp. Neurol. **39**:173, 1925.

38. Chu, H. N.: The Cell Masses of the Diencephalon of the Opossum Didelphis Virginiana, Academia Sinica, Monographs, serial no. 2, Peiping, National Research Institute of Psychology, 1932; Fiber Connections of the Diencephalon of the Opossum Didelphis Virginiana, Academia Sinica, Monographs, serial no. 3, ibid., 1932.

39. Ingram, W. R.; Hannet, F. L., and Ranson, S. W.: Topography of the Nuclei of the Diencephalon of the Cat, J. Comp. Neurol. **55**:333, 1932.

40. Winkler, C., and Potter, A.: An Anatomical Guide to Experimental Researches on the Cat's Brain, Amsterdam, W. Versluys, 1914.

In addition to these two well known nuclei, Fuse [41] discovered a gray mass which he called the "gewundenes Grau," or "Olivenkern des vorderen Vierhuegels" ("twisted gray," or "olivary nucleus of the superior colliculus"). According to the description, this convoluted nuclear mass belongs to the pretectal group in the wider sense of the word. Measuring 0.8 to 1.1 mm. in size, it is seen laterally in the stratum griseum intermedium, occasionally extending into the griseum

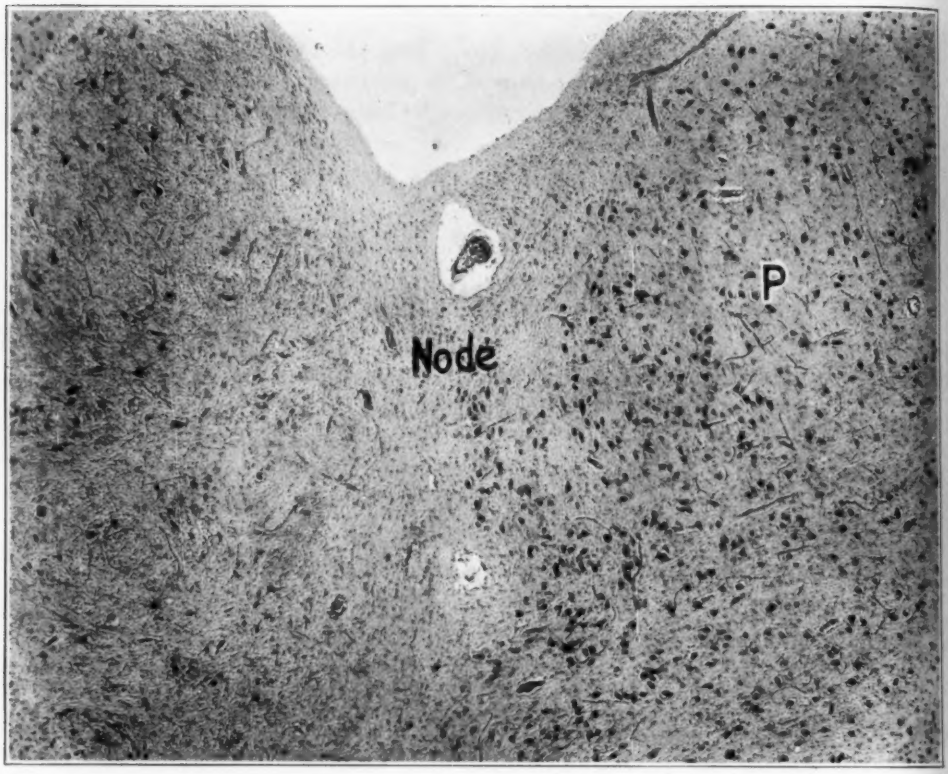

Fig. 8.—Dorsolateral optic nucleus of the midbrain (nucleus magnocellularis tractus optici [*Node*], LeGros Clark). Right, the pulvinar (*P*); left, posterior part of the habenular ganglion, with its large cells; between the two, below the vessel, the dorsolateral optic nucleus of the midbrain. (Cresyl violet.)

superficiale near the pulvinar. It is in the closest relation to the brachium of the superior colliculus. It was possible to identify this olivary nucleus of the superior colliculus as described by Fuse (fig. 7). It can be

41. Fuse, G.: Das gewundene Grau oder der Olivenkern des vorderen Zweihügels, Nucleus olivaris corporis quadrigemini arterioris bei Mensch und Tier, Arb. a. d. anat. Inst. d. kaiserlich-japan. Univ. zu Sendai, 1936, no. 19, p. 49.

identified also in man. It can be distinguished from the other nuclei by its clearer ground substance, resembling a gelatinous substance. According to Fuse, there are many fibers connecting this nucleus with other nuclei and also a direct connection from the retina. Its significance depends on its manifold connections. Its site precludes its being compared to the ventral nucleus of the lateral geniculate body or to the griseum parageniculatum, which cell masses it definitely resembles.

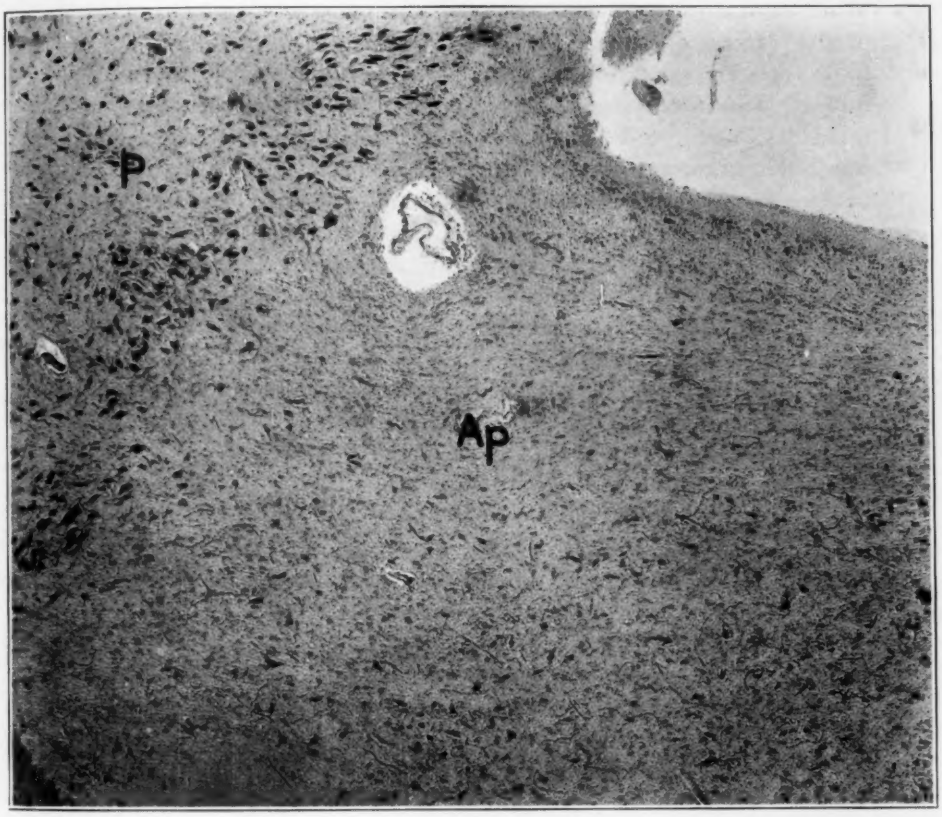

Fig. 9.—Area praetectalis in man (*Ap*). Left, nucleus limitans of the pulvinar (*P*); medioventral from the two blood vessels, the area; the larger cells more ventrolateral, the pretectal nucleus.

The last of the midbrain nuclei is the ganglion ectomammillare of Edinger, called by many authors the ganglion opticum tegmenti ventrale. Many years ago I [42] described this nucleus in various animals and in

42. Marburg, O.: Basale Opticuswurzel und Tractus peduncularis transversus, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **10**:1, 1903.

man. It was found that that part of the tractus peduncularis transversus which originates in the retina is to be compared with the basal optic root of birds, ending in the ectomammillary ganglion. It was a misunderstanding by Frey,[28] who examined this bundle a few years ago, when he stated that I had homologized the basal optic root with the tractus peduncularis transversus. Merely the opinion had been expressed that one part of this tract, namely that which originates in the retina, is identical with the basal optic root. This fact was also confirmed by Wallenberg,[43] Loepp [20] and many others. Frey [28] suggested a connection of this ventral optic ganglion to the oculomotor nucleus, as found in birds by Edinger [44] (Groebbels' [45] "Haubenbündel des Ectomammilare") and in other vertebrates by Perlia,[7] Shanklin [46] and others. I could not observe these fibers in birds as distinctly as Groebbels,[45] nor was I able to confirm their presence in the higher vertebrates.

However, fibers were seen in birds, well known to the older authors, which ran more laterally and ended in the ventral mesencephalic nuclei, entering perhaps also into the substantia nigra (Castaldi,[47] Sterzi [48]). There are also fibers in birds which directly connect the basal optic root as well as the ganglion ectomammillare with the medial part of the tectum. In man there are only fibers arising from or ending in the stratum zonale and partly in the stratum opticum of the colliculus, joining the tractus peduncularis transversus where it turns from the orocaudal direction into the lateromedial. Their exact nature cannot be revealed in normal preparations.

It may therefore be concluded that there are many nuclei in the midbrain, in connection with fibers from the retina, nuclei which would be better called "optic nuclei of the midbrain," namely the superficial gray matter of the tectum, which receives only a few noncrossed and more crossed retinal fibers (tractus retinotectalis). The large cells situated medially in this layer are probably equivalents of the magnocellular nucleus of the ganglion isthmi, with its tractus retinoisthmicus. This tract probably lies in the higher forms in the small medial optic

43. Wallenberg, A.: Notiz zur Anatomie des tractus peduncularis transversus beim Meerschweinchen, Anat. Anz. **24**:199, 1903.

44. Edinger, L., and Wallenberg, A.: Untersuchungen über das Gehirn der Tauben, Anat. Anz. **15**:245, 1899.

45. Groebbels, F.: Untersuchungen über den Thalamus und das Mittelhirn der Vögel, Anat. Anz. **57**:385, 1924.

46. Shanklin, W. M.: The Central Nervous System of Chameleon Vulgaris, Acta zool. **11**:425, 1930; The Comparative Neurology of the Nucleus Opticus Tegmenti with Special Reference to Chameleon Vulgaris, ibid. **14**:163, 1933.

47. Castaldi, L.: Studi sulla struttura e sullo sviluppo del mesencefalo: Ricerche in cavia cobaya, Arch. ital. di anat. e di embriol. **20**:23, 1923; **21**:172, 1924; **23**:481, 1926.

48. Sterzi, G.: Anatomia del sistema nervoso centrale dell' uomo, Padua, A. Draghi, 1914-1915.

root, but this fact remains to be proved. Laterally may be found the magnocellular nucleus of the optic tract (LeGros Clark [15]), to be seen also in man, which receives retinal fibers. I deem it better to give to these two nuclei the names "nucleus opticus mesencephali dorsalis medialis" and "lateralis"—dorsomedial and dorsolateral optic nucleus of the midbrain—in order to show by these names their significance, whereas the collicular olive of Fuse may remain so called until a better knowledge of it has been established. The ectomammillary nucleus, a primary end station of retinal fibers, represents a nucleus opticus mesencephali ventralis (ventral tegmental optic nucleus; fig. 6).

Concerning the function of all these various nuclei and pathways, it may be stated that only the lateral geniculate body in its dorsal portion carries light impulses to the calcarine cortex. All the other nuclei serve only reflex function. Except in the pupillary reflexes, they are of no great significance, because the subcortical optic relations of the retina are subordinated to the cortical relations. The higher organized the animal, the less significant are the subcortical centers. Therefore direct retinal connections to subcortical centers related to higher psychic functions (attention, colliculus, pulvinar; affect, medial nucleus of the optic thalamus) are of little value. That is seemingly true also of the hypothalamus (vasomotor centers). The only important direct connections are those for the pupillary reactions. It may be that the fasciculus retinosubthalamicus can be brought in relation to the tonic pupillary reflexes (Adie phenomenon), whereas three of the collicular optic nuclei (nucleus opticus mesencephali dorsolateralis, Fuse's collicular olive and the nucleus opticus mesencephali ventralis) are related to the pupillary light reflex. The investigations of Ranson and Magoun [49] make it likely that in the region of the posterior commissure and around the aqueduct there are cells or fibers carrying light impulses for the pupillary light reflex to the oculomotor nucleus. According to Lenz [50] these fibers are interrupted in the Darkshevich nucleus (nucleus commissurae posterior). But Spiegel,[51] having destroyed this region without resulting loss of the light reflex, suggested that nuclei or fibers connected with the reflex may be situated more laterally. If Loewenstein [52] is right that fibers carrying light impulses to the oculomotor

49. Ranson, S. W., and Magoun, H. W.: Respiratory and Pupillary Reactions Induced by Electrical Stimulation of the Hypothalamus, Arch. Neurol. & Psychiat. **29**:1179 (June) 1933; The Ventral Path of the Pupilloconstrictor Reflex in Response to Light, ibid. **30**:1193 (Dec.) 1933.

50. Lenz, G.: Untersuchungen über die anatomischen Grundlagen von Pupillenstörungen, insbesondere der reflektorischen Pupillenstarre, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **46**:234, 1929.

51. Spiegel, E. A.: Die Zentren des autonomen Nervensystems, Berlin, Julius Springer, 1928.

52. Lowenstein: Personal communication to the author.

nuclei are interrupted by other nuclei, only the previously mentioned two dorsal nuclei represent this interruption.

But in 1903 I [53] suggested that the ventral mesenecephalic optic nucleus may be in relation to the light reflex in the manner recently agreed on by Frey.[28] This comment is significant in showing how little is known about this simple phenomenon. It seems impossible to have a correct opinion about the isthmus ganglion or its equivalents in higher organized animals. The existence of its two parts, the one belonging to the cochlear and the other to the optic system, lend credence to the view that there is a simultaneous center of impressions from eye and ear with a direct influence on respective muscles.

This is as far as one can go in regard to the anatomic relations; physiology may fill in the gaps to show the character of the functional process made possible by these manifold connections.

SUMMARY

All the optic fibers arise in the retina. Antidromic fibers are absent. Apart from the retinogeniculate and pregeniculate fibers, those to the pulvinar (unmyelinated) are still under discussion. Greving's retinothalamic tracts are to be confirmed, as is the retinosubthalamic fasciculus. There are three retinohypothalamic tracts: the fasciculus retinosupraopticus, the fasciculus retinotuberis medialis and the fasciculus retinotuberis lateralis. Their respective nuclei are connected with the hypophysial gland (in the frog, uninterrupted—fasciculus retinohypophyseus).

The existence of a medial optic root in the midbrain of man could be claimed, as in lower-organized animals. Its connection to scattered cells medially in the tectum resembles the optic connection to the ganglion isthmi.

The nucleus magnocellularis tractus optici (dorsolateral mesencephalic optic nucleus) in man is a small circumscribed cellular group dorsal and anterior to Fuse's collicular olive.

The ventral mesencephalic optic nucleus (ganglion ectomammillare), the end station of the basal optic root, is connected with the tectum, into which enter in birds direct and indirect optic fibers of the basal root. In man the dorsal limb of the tractus peduncularis transversus reaches laterally the stratum zonale and the stratum opticum of the anterior colliculus.

Concerning the functions, the pathways of the pupillary light reflex, in particular, are discussed.

225 Central Park West.

53. Marburg, O.: Die diagnostische Bedeutung der Pupillenreactionen, Wien. Klin. **29**:235, 1903.

ALLERGIES IN OPHTHALMOLOGY

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True allergy is a hypersensitivity of tissue cells to allergens. The cells of practically every tissue of the body may acquire a hypersensitivity. This may come about through either systemic or local absorption of the allergen in a person in the proper biochemical or physiologic state. Likewise, in some persons nearly every tissue may develop an immunity. Most persons who become allergic inherit a tendency. Then infection, shock or emotional strain may precipitate the allergic state when the subject is exposed to allergens in sufficient quantities.

The various tissues of the eyes are no exception to the susceptibility to allergic manifestations.

No effort will be made to review the literature on allergies in ophthalmology, as that has recently been well done by Appelbaum.[1]

To understand better the allergies of the eyes it is necessary to have a good conception of allergies in general. From over 50 per cent of susceptible persons one can elicit a history of allergies in the immediate members of the family. The patient himself frequently gives a history of a dislike and intolerance of certain foods and of eczema, urticaria, asthma, hay fever, bronchitis, periodic headaches, vasomotor rhinitis and sinusitis. He usually has a slow pulse, low blood pressure, a spastic gastrointestinal tract, hypochlorhydria, a tendency to increased eosinophilia and lowered basal metabolism. The bronchial, vaginal, nasal and ocular secretions show an increase in eosinophils. The p_H of the conjunctival secretion and that of the tears are increased. Fabricant [2] found that the p_H of the nasal secretion is increased in allergy and that there is a parallel rhythmic variation in all mucous surfaces. Allergic patients are above average in intelligence, with a hyperstimulation of the parasympathetic nervous system or at least a more active parasympathetic than sympathetic system.

The biochemical examination of allergic patients reveals definite tendencies. The blood sugar content is usually low and the amino acid and the blood urea content high. The value for cholesterol is usually

1. Appelbaum, A.: Allergic Phenomena in Ophthalmology, Arch. Ophth. **24**: 803 (Oct.) 1940.

2. Fabricant, N. D.: The Significance of the p_H of Nasal Secretions in Situ, Tr. Am. Acad. Ophth. (1940) **45**:197, 1941.

in the upper normal range or slightly increased. The sodium, phosphorus and potassium vary with the state of the patient. Sheldon and associates [3] pointed out that during asthmatic attacks the loss of water and sodium through the urine was sometimes double the preasthmatic output, that the water metabolism was directly parallel to the sodium metabolism, that there was a proportionate loss of weight and that this was independent of the sodium-water intake. The loss occurred in spite of distinct edema of the shock organ tissues. This is explained by the fact that the kidneys excrete sodium and water and the potassium is reabsorbed by the tubules of the kidneys. The loss of sodium in the blood lowers the electrolytes and thereby draws the potassium from the tissues, resulting in a lowering of the tissue potassium (Parker [4]). If potassium is given it does not increase the blood potassium but increases the tissue potassium. The loss of water and sodium and not of potassium during an attack would then make the potassium level appear higher at that time.

It is known that the adrenal cortex controls the sodium-potassium-water balance; that is, with a deficiency of the secretions of the adrenal cortex there are loss of sodium and water and retention of potassium. It is also known that the adrenal medulla diminishes the edema of the shock organ tissues. Since in allergy there are a loss of sodium and water and edema of the shock organ tissues, since the adrenal cortex regulates the sodium-potassium-water balance and since the medulla reduces the edema of the shock organ tissues, it stands to reason that a dysfunction of the adrenal gland must play a great part in the disturbed sodium-potassium-water balance and in the edema of the shock organ tissues in allergic manifestations.

In allergic reactions the histamine concentration in the blood is increased, which is a factor in lowering the blood pressure. An excess of histamine in the blood is neutralized by histaminase, but the modus operandi is not fully understood. Acetylphenylhydrazine has also been thought to neutralize the excess of histamine in the blood.

The imbalance of the vegetative nervous system in allergic persons is an important factor and is affected by a variety of causes. Psychic disturbances occasionally are responsible. The pressure of modern city life, with insufficient physical activity, frequently throws the nervous balance on the side of overactivity of the parasympathetic system. The reason for hyperstimulation of the vagus is difficult to explain fully, but it is known that the posterior lobe of the pituitary gland, the adrenal

3. Sheldon, J.; Howes, H., and Stuart, G.: Observations on Total Water and Sodium Exchanges in Asthmatic Patients, J. Allergy **11**:1 (Nov.) 1939.

4. Parker, F. P.: Blood Potassium Studies in Allergic States: Relation to Potassium Therapy, South. M. J. **33**:1301 (Dec.) 1940.

medulla and the thyroid gland are sympathetic stimulants and that the adrenal cortex is a parasympathetic sedative, and it would seem that the hormones of these glands must be a factor in bringing about an imbalance of the vegetative nervous system. It is also known that many patients acquire their allergies or become immune at puberty or the menopause. Consequently, the gonads must also play a part in the altered biochemical metabolism or the neurologic state present in the various allergic conditions.

To summarize the role of the endocrine glands in allergy, it would seem that the allergic patient has lowered thyroid function, intermittent loss of sodium and water, edema of shock organ tissues, lowered blood pressure, parasympathetic hyperstimulation, a low blood sugar content and an increase of cholesterol, urea and amino acids. The thyroid gland diminishes the cholesterol, increases the blood sugar and diminishes edema. It is greatly dependent on the anterior lobe of the pituitary and on the adrenal gland for its function. The adrenal cortex prevents the loss of sodium and water from the blood and lowers the content of amino acids and urea and is a sedative for the vagus, and the medulla reduces the edema of the shock organs and stimulates the sympathetic system. The posterior lobe of the pituitary gland also diminishes the edema of the shock organ tissues, stimulates the sympathetic system and diminishes the loss of water. The adrenal cortex is entirely dependent on the anterior lobe of the pituitary gland for its function. However, fatigue, infections and toxemias are common causes of adrenal exhaustion even in the presence of a normal anterior lobe of the pituitary gland. Apparently the gonads, which are dependent on this lobe and on the adrenal glands for their function, also play a part; therefore, the key glands responsible for most of the findings in allergy must be the whole pituitary gland and the adrenal glands.

In a number of patients with allergy of long standing one may encounter an increase in the blood pressure and in the pulse rate, with many of the other findings diminished. Some of these patients become less allergic when such changes take place.

The aim of this essay is to discuss the clinical aspects of allergies of the eye; consequently, drugs and chemicals to which a patient may become hypersensitive will be included as well as true allergens.

LIDS

The skin of the lids is susceptible to the same allergic manifestations as are found in the skin of other parts of the body, but its reaction is more severe than that of most other cutaneous surfaces. Eczema of the lids, with its intense itching, burning, redness and thickening of the skin, may be due to chemicals or to drugs such as atropine, yellow oxide of

mercury ointment, metaphen ointment, merthiolate, scopolamine, homatropine, butyn, pontacaine, phenacaine and physostigmine. It may also be due to cosmetics, powder, face cream, mascara, finger nail polish, face lotion and dye on the lashes, brows or hair. It may be due to feathers in pillows, horsehair mattresses, kapok, fur coats or fur trimming on suits and white metal or zylonite spectacles. It may also be caused by ingested foods, by pollens and by plant exudates. I have seen eczema of the lids caused by each article mentioned. Blepharitis sicca and blepharitis ulcerosa are frequently due to allergy.

CONJUNCTIVA

Allergy of the conjunctiva may be due to the same causes responsible for allergy of the skin; in fact, the two are frequently present at the same time. The former is usually associated with burning and intense itching of the margin of the lids, especially at the nasal corner. For convenience in discussion it can be classified into four types: the acute type with chemosis of the bulbar conjunctiva, the acute type with redness and some follicular hypertrophy, the chronic type and vernal catarrh. In some cases of the acute type the chemosis may be so severe that the conjunctiva protrudes between the lids. There is profuse lacrimination and burning, with moderate congestion. These pathologic conditions are nearly all due to direct contact of the sensitized conjunctiva with a specific antigen, most commonly a pollen. The onset is sudden, and there is rapid clearing with use of neo-synephrin or epinephrine and cold compresses. From a theoretic standpoint heat diminishes the p_H of the secretions and cold increases it, but cold diminishes the swelling while heat increases it; so for the immediate results cold is best. In the second type of acute or subacute allergic conjunctivitis the onset is not so rapid; in fact, this type may come on rather slowly. There are follicles of the conjunctiva, most noticeable in the lower cul-de-sac, and occasionally they are severe, even partially covering the corneal limbus. The congestion may be extreme. The lower half of the eye is nearly always more congested than the portion under the upper lid. There are itching and burning, with more or less sticky, rubbery, mucoid discharge of a high p_H. This type is frequently associated with eczema of the lids. Usually it is due to drugs, cosmetics, air-borne animal dust, plant exudates or foods. The chronic type may go on for years with recurrent subacute exacerbations. Usually there are follicles during the exacerbations, but between times these may clear up, leaving only congestion and slight itching; the patient is scarcely ever entirely well. I have seen many such patients who return summer after summer, and they usually return on approximately the same date each year. As with the previous type of condition, the eye is whiter under the upper lid. This type is due to ingested foods, cosmetics

of all kinds and air-borne allergens, both animal and plant. It may be associated with low grade sinusitis and seem to be due to a bacterial allergen. It presents the most difficult problem to solve, as it is usually due not to a single allergen but to many, and on removal of a few of the provocative allergens the patient improves but does not get entirely well.

Since 1925 I have considered vernal catarrh as allergic in origin, although there still seems to be some disagreement as to the causation. My experience has only further confirmed the theory that it is an allergic manifestation. In 1925, at the American Academy of Ophthalmology and Otolaryngology, I reported several cases of vernal catarrh, and with but one exception all the patients were allergic to pollens prevalent during the season of the activity of the disease. The one who was not sensitive to pollen was sensitive to foods, with which the condition could be precipitated. Later I immunized several patients to the pollens to which they were hypersensitive, and the following summer they were practically free from the disease. The second summer it recurred in a moderate degree, and the third summer it was about as bad as it was before the artificial immunization. After finding that the immunity was so transitory, I became discouraged, feeling that the treatment was not justified, and I have never published those cases; but this experience confirmed my former opinion as to the causation of vernal catarrh.

In the 1925 paper I reported 4 cases of clinical trachoma with follicles, pannus and scarring of the conjunctiva, and I have noted several more since in which there were seasonal relapses during the pollinating season of the plant to which the patient was hypersensitive. These few cases do not prove anything, as the condition might have been trachoma in an allergic patient aggravated by the allergy or it might not have been true trachoma. I have not encountered such a case since the advent of sulfanilamide for the treatment of trachoma. However, it is well to have in mind that such cases occur. Trachomatous patients, like allergic patients, have a high p_H of the tears.

Phlyctenular conjunctivitis should be mentioned, as I consider it in most cases an allergic reaction to tuberculoprotein. In the few cases encountered in which it was not tuberculous it was due to other allergic factors, including sensitization to other bacteria.

CORNEA

Phlyctenular keratoconjunctivitis is generally regarded as an allergic manifestation, the most common etiologic factor being a sensitivity of the cornea to tuberculoprotein. In fact, in a patient who has had phlyctenular keratitis that has healed, an attack may be precipitated by instilling tuberculin into the eye. In about 10 per cent of the cases the disease is

due to bacterial toxins or to hypersensitivity to foods, but in these cases it is usually not as severe as when it is due to tuberculoprotein.

In nearly all cases interstitial keratitis is the result of congenital syphilis. In many cases spirochetes are in the cornea at birth, and apparently they sensitize the cornea. Later, when the patient has other active syphilitic foci the toxins of the spirochetes are liberated and set up an inflammation into any sensitized tissue. If the cornea has previously had spirochetes in it that sensitized it, interstitial keratitis develops. It is well to mention at this point that a number of investigators have found that patients with interstitial keratitis frequently have a deficient thyroid function.

A small percentage of interstitial keratitis is due to other causes, among which is tuberculosis, the underlying etiologic factor generally being considered a sensitivity of the cornea to tuberculoprotein, causing an allergic reaction. A few cases of interstitial keratitis which was the result of food or air-borne antigens have been reported.[5]

There is a variety of corneal ulcers that is allergic. I have seen pinpoint ulcers that nearly covered the whole cornea. They may be so small that they can be seen only by staining the cornea with fluorescein and then examining the eye with a slit lamp and corneal microscope. Then there is the superficial punctate ulcer around the periphery of the cornea. As I have reported before, typical dendritic ulcers may be precipitated by ingested food. I presume that ophthalmologists have all seen on the cornea near the limbus, at 5 to 7 o'clock, small ulcers that were due to medication. These are insignificant if the medication is discontinued, but if it is continued for some time they may progress to a point of irreparable damage. The proper care cannot be too strongly emphasized. In the past three years I have seen 5 patients in whom ointments and collyriums had caused ulcers that covered most of the cornea; 2 of these patients lost all useful vision, and the other 3 were left with greatly impaired vision. The antiseptic ointments are the most common offenders, although atropine and physostigmine may also cause the ulcers.

There are corneal ulcers caused by dyes used on the hair, brows and lashes; some of these are severe.

SCLERA

The sclera reacts to the same allergen as the cornea, but, being protected by the conjunctiva, it can be affected only by allergens in the circulation. I have had 2 cases of severe necrosing scleritis that seemed to be due to tuberculosis. In one the condition had existed for ten years

5. Dean, A. M.; Dean, F. W., and McCutchan, G. R.: Interstitial Keratitis Caused by Specific Sensitivity to Ingested Foods, Tr. Sect. Ophth., A. M. A., 1939, p. 381.

and after hygienic measures and tuberculin therapy the eyes completely quieted down. In the other there was also arthritis. The ocular condition had been getting progressively worse for two years. The patient in this case also was sensitive to tuberculin. The only addition to her former treatment was tuberculin, and in six months the eyes were quiet, the vision 20/30 with correction and the bulging choroid flattened out with scar tissue.

UVEAL TRACT

Much experimental work has been done that points to the probability that in many cases uveitis is allergic in origin.

The allergy is frequently due to the toxins of the tubercle bacillus or to the toxins of foci of infection.

The mild iritis with a subacute cold that clears up with only a mydriatic in three or four days is of allergic origin. I have seen patients with an increase in the cells in the anterior chamber, slight discoloration of the iris and a sluggish pupil associated with a severe systemic allergic reaction. No adhesion of the iris to the lens resulted in any of them, but there is a uveal involvement that is apparently due to the allergic phenomenon.

In 1910 Elschnig [6] advanced the theory that in sympathetic ophthalmia the uveal pigment of the injured eye acts as an antigen, causing a hypersensitivity of the body tissues to the pigment. This theory applies especially to the uveal cells of the fellow eye, and uveal pigment subsequently absorbed produces an allergic reaction in the pigmented tissues of the uninjured eye. This theory has been confirmed by Woods [7] and others, and to date it is the most widely accepted theory on the causation of sympathetic ophthalmia.

LENS

There have been reported in the literature a number of cases of cataract occurring in a patient with eczema and asthma. I have seen opacities of the lenses develop in a number of cases while the patient was having severe allergic manifestations, and in some cases the progress of the opacities subsided after the reaction ceased. The percentage of patients with presenile cataract having allergy has been high, and some of these patients have been sensitive to lens protein. The question that naturally arises is whether allergy is a causative factor in them. Since the lens is of ectodermal origin, it seems possible that in patients having eczema the lens might also be affected. Most of these patients have a

6. Elschnig, A.: Studien zur sympathischen Ophthalmie: II. Die antigene Wirkung des Augenpigments, Arch. f. Ophth. **76**:509, 1910.

7. Woods, A. C.: Ocular Anaphylaxis: III. The Role of Uveal Pigment, Arch. Ophth. **46**:283 (July) 1917.

deficient thyroid function. Beetham [8] pointed out that histologic examination of the crystalline lens showed cortical degeneration and a normal capsule. However, the lens fiber, being less resistant to degenerative change, might show changes from edema when the capsule was normal.

Verhoeff and Lemoine [9] in 1922 brought out the theory that intraocular inflammation following rupture of the lens capsule, traumatic, surgical or spontaneous, is due to an allergy to lens matter. They also described a typical histologic picture observed in five enucleated eyes. There was a positive cutaneous reaction to lens protein in all cases in which such an inflammation developed. About 8 per cent of all adult patients gave a positive cutaneous reaction to lens protein. In 1924 Lemoine and Macdonald [10] confirmed these findings. Since that time, in my practice they have been further confirmed but without confirmation of any severe endophthalmitis phacoanaphylactica, because when a patient with immature cataract was found to be hypersensitive to lens protein the intracapsular operation was performed, and this obviated the possibility of any remaining lens substance. A moderate inflammation was noted in a few cases of mature cataract in which operation was by the capsulotomy method. The inflammation varies directly with the degree of hypersensitivity and the amount of lens matter remaining in the eye. DeVeer [11] reported 3 cases in which there were the characteristic histologic changes of endophthalmitis phacoanaphylactica following rupture of the lens capsule, with ensuing sympathetic ophthalmia. He advanced the hypothesis that endophthalmitis phacoanaphylactica is occasionally a precursor of sympathetic ophthalmia. This opinion would seem to fit in with the Elschnig theory of sympathetic ophthalmia discussed under "Uvea."

MUSCLES

In 1925 I reported a case of diplopia due to the eating of cheese, to which the patient was hypersensitive. Since that time I have not encountered a similar case in which I was able to prove an allergic origin. A number of cases of transitory severe muscle imbalance have been observed, and patients with normal muscle balance, some of whom were allergic, have complained of transitory recurrent diplopia, but I have not been able to produce the condition at will. It is highly possible that in many of these cases the imbalance was allergic in origin.

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8. Beetham, W. P.: Atopic Cataracts, Arch. Ophth. **24**:21 (July) 1940.

9. Verhoeff, F. H., and Lemoine, A. N.: Endophthalmitis Phacoanaphylactica, Tr. Internat. Ophth. Cong., 1922, p. 234.

10. Lemoine, A. N., and Macdonald, A. E.: Observation on Phacoanaphylactica Endophthalmitis, Arch. Ophth. **53**:101 (March) 1924.

11. deVeer, J. A.: Endophthalmitis Phacoanaphylactica and Its Relation to Sympathetic Ophthalmia, Arch. Ophth. **23**:237 (Feb.) 1940.

RETINA AND OPTIC NERVE

Several authors have reported cases of retinitis as caused by allergy. There are so few such cases that it is difficult to draw definite conclusions. I have noted cases of retinitis in most of which there were multiple retinal hemorrhages of unknown origin that came while the patient had active allergic manifestations, but the course of the disease did not convincingly indicate an allergic origin except when there was a tuberculous causation. A diffuse edema of the retina extending 3 or 4 disk diameters around the optic nerve head and involving the macula lutea has frequently been observed. This condition was so severe in some cases that it reduced the vision to 20/50 and caused an elevation of the optic disk as much as 3 D. I at first considered the origin allergic, but later I noted the condition in patients in whom it was not possible to demonstrate an allergy. These patients were studied further, and they were found to have a deficient thyroid function. As many of those in whom the condition had previously been attributed to allergy were subsequently checked over for their thyroid function and all were found to be more or less of the hypothyroid type, I came to the conclusion that the primary cause of the edema described must be a deficient thyroid function, but the fact that most of the patients were allergic further associated the cause and the effect.

A number of authors have reported cases of detachment of the retina as allergic in origin. There is every reason to believe that their reports are correct, as one can easily visualize such a reaction in the retina. I have found no cases in which detachment could conclusively be attributed to allergy.

Retrobulbar neuritis could be attributed to allergy in some cases. This is especially true of the chronic type usually called toxic amblyopia. Some patients get toxic amblyopia from quantities of the toxic substance which are extremely small when compared with the average consumption or exposure of most people. Even retrobulbar neuritis attributed to multiple sclerosis could be attributed to allergy if the theory of multiple sclerosis advanced by some authors was accepted. I do not yet accept this theory on the etiology of multiple sclerosis.

TREATMENT

It is natural that the treatment of a condition such as allergy of the eyes, which may have so many underlying etiologic factors and may be due to such a variety of allergens, cannot be uniform in all cases.

In many cases a careful history is sufficient to point out the allergen, or allergens, responsible for the condition, and simple withdrawal from the surroundings or from the diet is all that is necessary. If this fails, if the condition is mild the average patient does not wish to go to the

expense necessary for an accurate diagnosis and should receive merely palliative treatment, such as irrigation with a slightly acid buffer. Many patients suffering from allergic reactions of the skin and lids due to food hypersensitivity are greatly benefited by histaminase in doses of 20 units three times a day.

In cases of severe allergic reactions, when it is impossible to determine the underlying cause or the precipitating antigen a thorough examination by an allergist and an internist is best. This examination should include a complete chemical examination of the blood, a Wassermann test, a tuberculin test if indicated and determination of the basal metabolic rate.

If the condition is found to be tuberculous, hygienic measures, vitamins, fresh air and rest are important. If the condition is severe, the patient should be immunized with tuberculin given preferably intradermally at intervals about weekly, depending on the length of the reaction following the treatment. Gold sodium thiosulfate in 5 to 10 mg. doses every two days frequently arrests the lesion.

If the condition is found to be due to syphilis, as in interstitial keratitis, fever therapy combined with chemotherapy is much the best form of treatment. The fever therapy stimulates the metabolic processes and the reticuloendothelial system, thus helping to overcome the disease as well as diminishing the allergy.

Patients found to have foci of infection should by all means have them removed, as in some patients who are sensitive to bacterial toxins the condition completely clears up after their removal. Frequently foci of infection aggravate the allergic reaction to other antigens, so that after their removal the patient is found to be less allergic to food, etc.

With patients who have severe allergic ocular conditions, who suffer from other allergic manifestations and who have a disturbed biochemical state and an endocrine imbalance, it is essential that this underlying disturbance be corrected. If then the condition does not clear sufficiently, it is best to immunize the patient to the offending allergen. This immunization is best carried out by an allergist.

As to the local treatment for eczema of the lids, topical application of 2 per cent silver nitrate solution to the cutaneous surface and use of a mild ointment, such as zinc oxide ointment, to keep the skin soft are advisable. The silver nitrate solution is also used for blepharitis. For allergic conjunctivitis all astringents and antiseptics should be avoided. Lotions with a low p_H should be used. Cold compresses give much relief. If the manifestations are severe, 0.25 per cent neo-synephrin hydrochloride solution or 1:2,000 epinephrine hydrochloride solution should be used. Neo-synephrin and epinephrine lower the p_H more than other vasoconstrictors and tend to lower the p_H of the tears. This observation conforms to the findings of Fabricant [2] concerning the nasal secretions.

Occasionally it is necessary to use a local anesthetic, but the fact should never be lost sight of that the patient may have or acquire a sensitivity to the anesthetic. Colored glasses worn when the patient is in bright light give considerable relief.

If for financial reasons it is necessary for the ophthalmologist to take care of the medical problem, until more is understood about the biochemical, neurologic and physiologic aspects of allergy a rational therapeutic measure for patients with low blood pressure is to give one or two 2 grain (0.13 Gm.) doses of adrenal concentrate daily and a small dose of thyroid, while patients who have a higher pulse rate and an increase in blood pressure should receive only adrenal cortical extract in 3 grain (0.19 Gm.) doses twice daily. Some patients do well with calcium. Afenil given intravenously is better than other preparations when the condition is stubborn.

Patients with immature cataract who are found hypersensitive to lens antigen should be operated on by the intracapsular method. If it is deemed inadvisable to operate by this method the patient should be immunized to lens protein and then operated on by the ordinary method. The full dose of lens antigen should be given at weekly intervals while the patient is convalescing from the operation.

CONCLUSIONS

The underlying cause of the disturbed biochemical and neurologic state of patients with allergy seems to be an impaired function of the pituitary and adrenal glands.

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DISCUSSION

DR. CHARLES A. BAHN, New Orleans: Many ophthalmologists are allergic to the allergies. In the many complexities of this subject, they easily become confused in a maze of unimportant details and forget to think in terms of simple but important fundamental principles. These principles largely determine the correct type of treatment. Notwithstanding the thoroughness and the timeliness of Dr. Lemoine's presentation, several phases of the subject of allergy need amplification.

One thinks and speaks of allergic diseases as though they have a single all-inclusive cause. This is seldom, if ever, true. Not one but an integration of three basic causative factors in varying proportions are responsible for the group of symptoms that are called allergic manifestations. These components are: (1) the prenatal factor, (2) the biologic time factor and (3) the environmental factor.

The sooner physicans learn to think in terms of the three dimensional causation of allergic diseases, the sooner will they be able to evaluate intelligently the technical details in the order of their importance.

By the first, or the prenatal, factor in allergy is meant the tendency to which some or all of the fixed body cells in the eye or other parts of the body are predisposed to increased sensitivities. This abnormal

excitability usually consists of definite patterns, and its manifestations depend on combinations with the second and the third factor, namely the biologic time factor and the environmental factor.

By the biologic time factor is meant the tendency of the fixed body cells to become altered during the various cycles of a person's life. Potentially, like the other factors, these alterations occur during a lifetime. With each succeeding generation of fixed body cells, especially in persons who are prenatally predisposed to certain allergies, the tendency to sensitivities is further augmented by the environment. During the transitional periods of life, such as infancy, adolescence, puberty, pregnancy, the climacteric and senility, these reactive changes are most pronounced.

By the environmental factor is meant the physical, chemical and psychic components, some of which have been mentioned by the essayist. The physical component includes mechanical force, heat, light and electricity. The chemical component includes organic and inorganic substances which are animal, vegetable and mineral in origin. Although psychic reactions are essentially physical or chemical, their importance in relation to allergy apparently justifies separate mention. According to Cook, approximately 10 per cent of the entire population of the United States (in other words, 13,000,000 persons) have definite allergic manifestations. Cells which have a predisposition to certain allergies, and as a result tissues as well, react abnormally to particular environmental stimuli. A person whose cells are not predisposed to the same allergies will experience no abnormal reactions from such environmental stimuli.

Allergic manifestations in patients suffering from ophthalmic disease are far more frequent and varied than is generally believed. These manifestations include the rarer entities which have a pronounced allergic component, such as angioneurotic edema, vernal conjunctivitis and sympathetic ophthalmia. Much more frequent, and of greater practical concern, is the predisposition to allergies of patients suffering from the more common conditions, such as subacute and chronic blepharitis, conjunctivitis and uveitis. When an allergic manifestation is associated with any of these conditions, healing becomes complicated and delayed. The parasitic, traumatic or metabolic features alone or together do not explain the patient's delayed or imperfect recovery from his ocular complaint. In at least 10 per cent of patients suffering from ocular conditions allergy should be considered as a possible entity.

There is a close relationship between vasomotor neuroses and allergic manifestations. The prenatal factor, the biologic time factor and the environmental factor must be considered in both these groups of conditions, and both consist of essentially abnormal vasomotor reactions to environmental conditions to which ninety of every one hundred persons easily adjust themselves.

Even a brief discussion of the local and general treatment of allergies of the eye is impossible here. Irrespective of the treatment administered to allergic patients, there is one important fundamental principle that should be heeded. This is the need of a hygienic routine intelligently directed and designed to minimize oversensitivenesses throughout the body by the common sense application of the fundamental principles of health. The routine should be adapted to the needs of each individual patient and is of especial value during the inactive and prodromal phases

of allergy. It involves an integration and budgeting of basic health essentials in their proper amounts on a definite time schedule, preferably based on the week. These fundamental essentials include: (1) work, (2) eating, (3) sleeping, (4) exercise, (5) recreation, (6) protection against excesses, (7) elimination, (8) cleanliness, (9) outlook on life and (10) the use of stimulants. Based on the one hundred and sixty-eight hours which constitute a week, the routine would average for (1) work, thirty-five to fifty-five hours; (2) eating, fifteen hours; (3) sleeping, fifty-five hours; (4) exercise, five hours; (5) outdoor recreation, ten hours. Approximately twenty-eight hours a week are thus left in which the patient can do as he desires.

In budgeting the average working day in a simple, practical way which is adaptable to the large majority of allergic patients no one schedule can best be assigned for the needs of all patients. On awakening, the patient takes three to four glasses of water (warm in winter, cool in summer). Then he does five to ten minutes of body-building exercises. He should begin preferably with five of any exercises which bring into action more or less uniformly practically all the muscles of the body. Each exercise should be repeated five times and increased by one more time daily until it is repeated twenty or twenty-five times. After exercising, the patient should take a cool bath, which should be followed by a brisk three minute body rub. A nourishing breakfast, consisting of fruit, cereal and meat or its equivalent is advisable. The day's maximum energy is best maintained by an adequate breakfast. Then comes the work of the morning. At noon a full hour should be allowed if possible; during the first half hour a light meal should be eaten and during the last half hour the patient should rest and relax. Then comes the work of the afternoon and evening, but this should not be extended so that the scheduled number of working hours per week is exceeded. For the evening meal the patient should eat as much of the food that agrees with him as he wishes. The allergic patient should avoid foods, fumes, dusts and everything else to which he knows he is allergic. The following regulations for the use of stimulants are important: The patient should not smoke until after the noon meal, and he should not smoke more than ten cigarets in any one day. The average number of cigarets should be about five per day. He should limit his consumption of caffeine to 2 grains (0.13 Gm.) per day, that is two cups of tea, coffee or other beverages containing caffeine. If the intake of alcohol helps to digest the evening meal, the limit should be two drinks in any one day, and the average should be one. For the patient who works indoors, a minimum of ten hours of outdoor recreation weekly is essential, preferably in not less than three instalments. I shall not discuss matters concerning protection against excesses of temperature, moisture, light and air pressure; elimination, cleanliness; outlook on life, and other details, because the time for this discussion is limited.

In my experience with the advocation of health routines for many more than 1,000 patients suffering from allergic disease, the necessary history requires about fifteen minutes and the instruction requires about the same length of time. Any ophthalmologist who will make use of health routines intelligently as an auxiliary in the treatment of allergic and other functional ocular diseases will soon find that the time and effort involved are well spent.

In conclusion, I hope that Dr. Lemoine's contribution attracted sufficient interest to cause allergy to be considered as a probable factor in ocular conditions which do not respond to treatment satisfactorily and in which the cause of the complaint and the effect of the treatment are not in proportion. The time and the effort spent will be more than justified.

DR. W. A. MARTIN JR., Atlanta, Ga.: I should like to mention potassium gluconate for the symptomatic relief of allergic conditions. This is put up in 7½ grain (0.48 Gm.) tablets. I usually start the treatment by giving five or six tablets three times a day. As soon as the symptoms are relieved, I gradually reduce the dose. In a great many cases one tablet three times a day will prove sufficient for an indefinite period.

DR. ALBERT N. LEMOINE, Kansas City, Mo.: I want to thank Dr. Bahn for calling attention to the extreme importance of hygienic measures. I brought that out but I am glad that he emphasized it, because it is one of the most important phases of the treatment of patients suffering from allergic conditions. The regulation of exercise and the relief of nervous tension are especially significant. Many patients become greatly improved or are relieved completely after a vacation in the country and a rest. Their extreme allergy usually subsides.

Ophthalmologic Reviews

EDITED BY DR. FRANCIS HEED ADLER

ACUTE METASTATIC GONORRHEAL DACRYOADENITIS

A CLINICAL AND HISTOLOGIC STUDY

J. M. RICHARDSON, M.D.

CHICAGO

OUTLINE

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III. Comment

IV. Summary and conclusions

I. LITERATURE

A. HISTORY

Knowledge of disease of the lacrimal apparatus is recorded in the earliest medical literature. Hirsch [1] found reference to lacrimal disease

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1. Hirsch, A.: Geschichte der Ophthalmologie im Alterhume, in Graefe, A., and Saemisch, E. T.: Handbuch der gesammten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1877, vol. 7, chap. 14, pt. 5, pp. 235-382.

in the Ebers Egyptian Papyrus of 1500 B. C. Schirmer [2] found seventy-three references to it between 1659 and 1800. Early nineteenth century writers on the subject include Schmidt [3] (1803), Beer [4] (1813) and Weller [5] (1821). Schmidt's treatise on the diseases of the lacrimal gland is an extensive work of value chiefly for its historical interest. He was the first to designate inflammation of the lacrimal gland as dacryoadenitis. Some of the etiologic factors named by him for inflammation of the lacrimal gland are still considered likely causes: (1) changes in the temperature of the air, (2) a scrofulous diathesis and (3) cold water.

The first case reports of diseases of the lacrimal gland were by Todd.[6] The conditions included inflammatory as well as neoplastic diseases. His description of the symptoms of acute dacryoadenitis is vivid and complete.

In spite of its antiquity, acute dacryoadenitis is considered a rare occurrence in reports in the literature and in textbooks. The textbook of Schmidt [3] is a notable exception. Arlt [7] up to 1853 had seen no patient with dacryoadenitis. Schirmer [2] in all his experience never saw one with acute dacryoadenitis. Seeligsohn [8] encountered 2 cases among 24.000 cases of ocular disease and Busacca [9] 3 cases among 30,000. Salvati [10] had the unique experience of observing 14 cases in ten years. Stock [11] among 70,000 cases of ocular disease observed only 5 cases of acute dacryoadenitis, in all of which the condition was unilateral. Up to 1925 he had seen no patient with bilateral involvement. Nicolau [12] gathered 254 cases of acute dacryoadenitis from the literature up to 1933.

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2. Schirmer, R.: Erkrankungen der Tränenorgane, in Graefe, A., and Saemisch, E. T.: Handbuch der gesammten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1877, vol. 7, chap. 12, pt. 5, pp. 1-45.

3. Schmidt, J. A.: Ueber die Krankheiten des Tränenorgans, Vienna, J. Geistinger, 1803.

4. Beer, G. J.: Lehre von den Augenheilkunde, Vienna, Camesina, Huebner u. Volke, 1813.

5. Weller, C.: Manual of Eye Diseases, Glasgow, Reid and Henderson, 1821, p. 195.

6. Todd, C. H.: Diseases of the Lacrimal Gland, Dublin Hosp. Rep. **3**:403-429, 1822.

7. Arlt, C. F.: Die Krankheiten des Auges, Prague, F. A. Credner & Kleinbub, 1853.

8. Seeligsohn, W.: Zwei Fälle von Dakryoadenitis, Klin. Monatsbl. f. Augenh. **30**:25-28, 1891.

9. Busacca, A.: Sulle dacrioadeniti acute, Rassegna ital. d'ottal. **2**:226-258, 1933.

10. Salvati: Contribution à l'étude de la dacrioadénite aiguë, Bull. Ophth. Soc. Egypt **25**:157-168, 1932.

11. Stock, W.: Pathologie der Tränendrüse, in Graefe, A., and Saemisch, E. T.: Handbuch der gesammten Augenheilkunde, Berlin, Julius Springer, 1925, vol. 9, chap. 15, pp. 34-48.

12. Nicolau (Mme.): Contributions à l'étude de la dacryoadénite aiguë primitive epidemique, Ann. d'ocul. **170**:722-778, 1933.

B. CLINICAL PICTURE OF ACUTE DACRYOADENITIS

Little space is given in the English textbooks to a description of the clinical course of acute dacryoadenitis. The following description is compiled from reports by Schirmer,[2] Todd,[6] Pes,[13] Wiener,[14] Stock[11] and Schieck and Brückner,[15] from the American Encyclopedia and Dictionary of Ophthalmology[16] and from personal observation. The condition begins with a sudden feeling of tenseness or even mild pain in the region of the lacrimal gland. This is followed by discoloration over the region and in a few hours by edema of the outer third of the upper lid. Pain, redness and swelling of the lid, gland and outer half of the conjunctival fornix progress rapidly. The swelling causes a droop of the temporal half of the upper lid. The S-shaped curve which this gives to the upper lid is characteristic of dacryoadenitis (fig. 1). The

Fig. 1.—A patient with acute dacryoadenitis (unilateral), showing the S-shaped curve of the margin of the upper lid (from Wiener[14]).

swelling usually continues to complete closure of the lids. The pain at this time is often exquisite and is made worse by motion of the lids and by pressure. On eversion of the upper lid, the gland and the conjunctiva appear as a red, swollen, tongue-shaped mass. Eversion is often impossible because of the severe pain. If the swelling continues

13. Pes, O.: (*a*) Doppelseitige akute Dakryoadenitis im Verlaufe einer akuten Gonorrhoe, Ophth. Klin. **3**:240-241, 1898; (*b*) Die acute bilaterale Entzündung der Tränendrüse bei Blennorrhagie der Urethra, Arch. f. Augenh. **51**:144-171, 1904.

14. Wiener, M.: Metastatic Dacryoadenitis in Gonorrhea, Quart. Bull. M. Dept. Washington Univ. **7**:35-39, 1909.

15. Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 7, pp. 371-376.

16. Dacryoadenitis, in Wood, C. A.: American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1914, vol. 5, pp. 3708-3709; Ocular Relations of Gonorrhea, ibid., 1915, vol. 7, pp. 5606-5613.

the gland may appear below the border of the upper lid. The chemosis is usually confined to the upper retrotarsal fold but may extend to the bulbar conjunctiva. When this happens the upper temporal quadrant is most often involved and the swelling may progress to the point where the bulbar conjunctiva folds over the upper half of the cornea. The vessels in the bulbar conjunctiva are at first only injected, but the palpebral conjunctiva becomes intensely hyperemic. There is at first epiphora, and this is followed by a mucoid discharge. The discharge may become purulent, the condition simulating purulent conjunctivitis. This description applies when the locus of inflammation is in the palpebral portion of the gland. When the orbital portion is involved the inflammatory processes combine to produce proptosis with diplopia. This may be confused with orbital cellulitis. There may be preauricular lymphadenitis at the height of the disease. In acute dacryoadenitis, malaise, fever, at times chills and even delirium may occur. In most cases the course is rapid but intense and there are no sequelae. Healing is complete in from a few days to three weeks. The condition may develop a subacute stage which heals slowly in from one to three months. The disease may terminate in one of three ways: (1) by complete healing, (2) by the formation of lacrimal cysts or dacryops or (3) by suppuration, with or without rupture. Cases of dacryops following dacryoadenitis were reported by Bialette [17] and Sourdille.[18a] Dacryops from any cause is rare (Seidel [19]). Suppurative dacryoadenitis is the least common form. Hirschberg [20] found only 1 case in a collection of 22,500 cases of ocular disease. Cases of dacryoadenitis which terminated in suppuration have been reported by Byers,[21] Stock,[11] Étiévant [22] and Sourdille.[18b] Sidler-Hugenin [23a] reported 1 case of his own (1911) and 1 of Haab (1887) in which an abscess formed in the outer third

17. Bialette, C.: Sopra un caso di cisti delle ghiandole acino tubulari, Atti r. Accad. di med. di Torino **4**:189, 1897.

18. Sourdille, G.: (*a*) Les tumeurs kystiques bénignes de la glande lacrymale (dacryops), Soc. franç. d'opht. **17**:244, 1899; (*b*) Des abscès chauds de la glande lacrymale palpébrale de Rosenmüller, Arch. d'opht. **20**:433-449, 1900.

19. Seidel, E.: Krankheiten der Tränendrüse, in Henke, F., and Lubarsch, O.: Handbuch der speciellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1931, vol. 11, chap. 2, pp. 281-347.

20. Hirschberg, J.: Mumps der Tränendrüsen, Arch. f. Augenh. **8**:186-187, 1897.

21. Byers, G. M.: Acute Suppurative Dacryoadenitis, Tr. Am. Ophth. Soc. **13**:135, 1912.

22. Étiévant: Dacryoadénite blennorrhagique, Lyons méd. **100**:485-487 and 509-513, 1903.

23. (*a*) Sidler-Hugenin: Concerning Metastatic Inflammation of the Eye in Gonorrhea, Arch. Ophth. **41**:360-376, 1912. (*b*) Haab, cited by Sidler-Hugenin. (*c*) Schaeffer-Buchardt, cited by Sidler-Hugenin.

of the right upper lid. In both cases there was spontaneous rupture, in Haab's case into the conjunctival sac and in the other externally. It is not clear from the description that these were not cases of dacryoadenitis. Gonococci were found in the pus in each case.

C. ETIOLOGY OF ACUTE DACRYOADENITIS

The causes of acute dacryoadenitis are varied. Injury was given as a cause by Bock [24] and Sgrosso.[25] Cases of acute dacryoadenitis due to cold were reported by Schmidt [3] and Elschnig.[26a] Rheumatic joint infection was given as a cause by Valude.[27] Acute dacryoadenitis following tonsillitis was described by Key [28] and Panas.[29] Cases of the disease due to influenza were described by Pignatari [30] and Nicolau.[12] Recurrent dacryoadenitis coincident with menstruation was reported by Lagrange and Armaignac.[31] Cases of dacryoadenitis due to scarlet fever and to measles were described by Bock.[24] Cholemia was given as a cause by Sgrosso.[25a] Dacryoadenitis due to erysipelas was described by Stock.[11] Cases of inflammation of the lacrimal gland due to a chemical cause were reported by Pes,[13] Prioux [32] and Grunert.[33] Acute miliary tuberculosis causing dacryoadenitis was described by Axenfeld.[34a] Focal infection was given as a cause by Foster.[35] Mumps causing dacryo-

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24. Bock, E.: Zur Kenntnis der gesunden und kranken Tränendrüse, Vienna, J. Šafář, 1896.

25. Sgrosso, P.: (*a*) Contributo alla dacrioadenite acuta, Ann. di ottal. **19**:159-165, 1890; (*b*) Dacrioadeniti del blennorhagia, ibid. **28**:78, 1898.

26. Elschnig, A.: (*a*) Ein Fall der akuter Dakryoadenitis, Centralbl. f. prakt. Augenh. **15**:353-357, 1891; (*b*) Gonorrhoe des Auges, in Finger, E.; Jadassohn, J.; Ehrmann, S., and Gross, E.: Handbuch der Geschlechtskrankheiten, Vienna, A. Hölder, 1912, vol. 2, pp. 67-80.

27. Valude, E.: Dacrioadénite rheumatique, Rev. gén. d'opht. **15**:179, 1896.

28. Key, B.: Bilateral Dacryoadenitis, Arch. Ophth. **50**:465-466, 1921.

29. Panas, P.: (*a*) Dacryoadénite double d'origine amygdalienne, Semaine méd. **3**:75-77, 1895; (*b*) Dacryoadénite, iritis et conjonctivite séro-vasculaire d'origine blennorrhagique, Rev. gén. de clin. et de thérap. **8**:169-171, 1894.

30. Pignatari, F.: La dacryoadénite consécutive à l'influenza, Rev. gén. d'opht. **13**:11-15, 1894.

31. Lagrange and Armaignac, H.: Dacryoadénite aiguë périodique et dépendant de la menstruation, J. de méd. de Bordeaux **30**:50-51, 1900.

32. Prioux, C.: Akute Dakryoadenitis nach kleinen Jodkaligaben, Ophth. Klin. **7**:19, 1903.

33. Grunert, K.: Augensymptom bei Vergiftungen mit Paraphenylendiamin, Ber. ü. d. Versamml. d. ophth. Gesellsch. **31**:208-217, 1902.

34. Axenfeld, T.: (*a*) Miliare Tuberkel der Tränendrüse, Ber. ü. d. Verhandl. d. IX internat. Ophth.-Kong., Utrecht, 1899; Ztschr. f. Augenh. (supp.) **2**:17, 1899; (*b*) Bemerkungen zur Physiologie und Histologie der Tränendrüse, Ber. ü. d. Versamml. d. ophth. Gesellsch. **27-28**:29-32, 1899.

35. Foster, M. L.: Acute Dacryoadenitis Due to Focal Infection, Arch. Ophth. **54**:430-431, 1925.

adenitis was reported by Hirschberg.[20] Pneumococcus was the cause ascribed by von Krudener [36] and Beauvieux.[37] Busacca [9] described cases of the disease due to the symbiosis of pneumococci and colon bacilli. Primary syphilis was given as a cause by de Lapersonne.[38] Diphtheria was the etiologic factor given by Pooley [39a] and Herken.[40] Anargyros [41] described dacryoadenitis due to dengue fever. Typhoid was the cause in cases reported by Lindner [42] and Radnot.[43] A case of dacryoadenitis due to the fusiform bacillus was described by Chamberlin.[44]

D. GONORRHEAL DACRYOADENITIS

The first case of dacryoadenitis in association with gonorrheal urethritis was reported by Miller [39b] in 1884, although he made no attempt to establish a connection between the two conditions. This was in discussion of a case of acute dacryoadenitis reported by Pooley [39a] at a meeting of the American Ophthalmological Society. The next report of dacryoadenitis in a patient with gonorrheal urethritis was by Seeligsohn.[8] He only suggested a possible etiologic connection between the gonorrhea and the inflammation of the lacrimal gland. He considered the possibility of metastasis. The first case report of dacryoadenitis definitely ascribed to gonorrhea was made by Panas [29b] in 1894. Only 39 cases of gonorrheal dacryoadenitis could be found in the available literature. These are summarized in the accompanying table. They include cases (*a*) in which no connection between the gonorrhea and the dacryoadenitis was established, (*b*) in which the authors assumed that the dacryoadenitis was due to other factors but in which from the history and the clinical course it most likely was due to gonorrhea and (*c*) in which the cause of the dacryoadenitis was stated to be gonorrhea.

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36. von Krudener, H.: Ueber Erkrankungen der Tränendrüse, Ber. ü. d. Versamml. d. ophth. Gesellsch. **31**:71-78, 1903.

37. Beauvieux: De la dacrioadénite palpébrale aiguë, Arch. d'opht. **29**:772-783, 1909.

38. de Lapersonne, F.: Dacryoadénites syphilitiques, Arch. d'opht. **22**:760-767, 1902.

39. (*a*) Pooley, T. R.: A Case of Acute Dacryoadenitis, Tr. Am. Ophth. Soc. **3**:729, 1884. (*b*) Miller, H. G., in discussion on Pooley.

40. Herken, H.: Beitrag zur pathologischen Histologie der Tränendrüsen, Arch. f. Augenh. **110**:61-75, 1937.

41. Anargyros, E.: Quelques cas de complications oculaires pendant l'épidemie de dengue, Arch. d'opht. **46**:214-216, 1929.

42. Lindner, S.: Ueber die Behandlung einiger Augenerkrankungen nach erloschener Influenzie-Epidemie in Wien, Wien. med. Wchnschr. **41**:681-682, 1891.

43. Radnot, M.: Die pathologische Histologie der Tränendrüse, in Supplementa ad Ophthalmologica, Basel, S. Karger, 1939, p. 28.

44. Chamberlin, W.: Subacute Dacryoadenitis with Conjunctivitis Due to Fusiform Bacilli, Arch. Ophth. **24**:602-604 (Sept.) 1940.

In the first series are the cases of Miller [39b] and Seeligsohn.[8] In the second are those of Key,[28] Maklakow [45] and de Lapersonne.[38] Key's case was an instance of acute bilateral dacryoadenitis in a 16 year old

Cases of Gonorrheal Dacryoadenitis Reported in the Literature

Author	Date	Patient's Age, Yr.	Patient's Sex	Type of Involvement	Duration of Dacryoadenitis	Duration of Gonorrhea	Complications
Miller [39b]	1884	..	..	Bilateral	3 days	Not given	
Seeligsohn [8]	1891	33	♂	Bilateral	2 mo., 28 days	6 mo.	Relapse
	1891	21	♂	Bilateral	9 days	6 mo.	
Panas [29b]	1894	"Young worker"	♂	Bilateral	Not given	6 wk.	Arthritis, metastatic conjunctivitis, keratitis
Gonella [47]	1898	20	♀	Bilateral	Not given	Not given	Arthritis
Pes [13]	1898	20	♂	Bilateral	5 days	"Acute" 1 wk.	
Sciememi [50]	1898	30	♀	Bilateral	Not given	Not given	
Sgrosso [25b]	1898	"Young student"	♂	Unilateral	"Healed quickly"	Not given	Suppuration
Terson [51]	1900	38	♂	Bilateral	1 mo.	1 mo.	
Maklakow [45]	1901	39	♀	Unilateral	1 mo.	2 yr.	Periglandular abscess
Ferry and Morax [48]	1902	25	♂	Bilateral	Not given	1 mo.	Arthritis, sclero-conjunctivitis, iritis
Grimaldi [52]	1902	23	♀	Bilateral	1 wk.	"Short time"	
de Lapersonne [38]	1902	..	♂	Bilateral	Not given	20 yr.	Mastitis, parotitis, iritis, epididymitis
Étiévant [22]	1903	22	♂	Bilateral	10 days	3 yr.	Suppuration
	1903	28	♂	Unilateral	2 wk.	2 mo.	
Cause [53]	1904	25	♂	Bilateral	2 wk.	Not given	
Pes [13]	1904	23	♂	Unilateral	4 days	3 yr.	Glottic edema, adenitis
	1904	21	♂	Bilateral	6 days	20 days	
	1904	57	♂	Bilateral	1 mo.	3 yr.	
Casali [54]	1906	26	♂	Bilateral	8 days	10 yr.	
Giani [55]	1906	"Junior officer"	♂	Bilateral	"Healed gradually"	10 days	Convulsions
Green [56]	1907	..	♀	Bilateral	Not given	6 mo.	Conjunctivitis
Wiener [14]	1909	20	♂	Unilateral	23 days	1 yr.	Preauricular and submaxillary lympnadenitis
Lindahl [57]	1910	..	..	Unilateral	Not given	Not given	Swelling of nasal mucosa
	1910	..	..	Bilateral	Not given		
Key [28]	1921	16	♀	Bilateral	Not given	3 mo.	
Romer [58]	1928	..	..	Bilateral	Not given	Not given	Arthritis
Vujtečh [40]	1929	..	..	Bilateral	Not given	Not given	Arthritis
Panico [59a]	1930	17	♀	Bilateral	12 wk.	1 yr.	Arthritis
de Petri [60]	1932	(2 cases; abstracted)					
Salvati [10]	1932	32	♂	Bilateral	3 wk.	12 days	
	1932	18	♂	Bilateral	2 wk.	1 mo.	
	1932	20	♂	Bilateral	3 wk.	1½ yr.	
	1932	29	♂	Bilateral	2 wk.	3 mo.	
	1932	21	♂	Bilateral	18 days		
	1932	35	♂	Bilateral	6 wk.	2 mo.	Arthritis
	1932	35	♂	Bilateral	2 wk.	2 yr.	
Dollfus [61]	1938	(article on treatment; no clinical details)					

girl who had had acute tonsillitis three weeks previously and gonorrhea three months before. At the time of the dacryoadenitis there was mild urethritis. Key stated the belief that the condition was "lacrimal

45. Maklakow, A.: Dacryoadenitis mit Bildung eines periglandularen Abscesses, Ophth. Klin. **5**:355-357, 1901.

mumps," but in view of the concurrent urethritis it is just as likely that it was of gonorrheal origin, activated or aggravated by the tonsillitis, which, even, could have been due to gonorrhea (Foster [46]). The first case of de Lapersonne [38] was of acute bilateral painful dacryoadenitis in a man whose Wassermann reaction of the blood was positive. There were painful epididymitis, parotitis and mastitis. The patient had had a gonorrheal infection with orchitis twenty years previously. Because of concurrent iritis and because of improvement in response to antisyphilitic treatment de Lapersonne stated the belief that the infection was syphilitic. It is improbable, however, that secondary or tertiary syphilis could have caused such acute painful adenitis, parotitis, mastitis and epididymitis. Gonorrhea is more likely to have caused the condition. So-called metastatic syphilitic lesions are almost never acute, and pain is "notable for its absence." De Lapersonne considered gonorrhea as a possible cause only to rule it out. Maklakow [45] could not account for the etiologic factor in his case but suggested atypical influenza. In the cases of Sidler-Hugenin [23a] and Haab [23b] the inflammation was due to gonorrhea, but whether the inflammation was in the lacrimal gland remains a matter of conjecture.

The cases in the third series, those in which the condition was ascribed to gonorrhea, are of two types: (*a*) those in which the lacrimal gland was involved by direct extension from infection of the conjunctival sac and (*b*) those in which the lacrimal gland was involved by metastasis. In the cases reported by Panas,[29b] Gonella,[47] Ferry and Morax [48] and Vujtečh,[49] purulent conjunctivitis, from the discharge of which the gonococcus was recovered in some instances, was followed by dacryoadenitis. In each of these cases, however, there was evidence of metastatic gonorrhea in other parts of the body, such as iritis, keratoiritis and arthritis. Because of this, Elschnig [26b] felt that involvement by direct extension from the conjunctival sac could be discounted and that in these cases the origin must be ascribed to metastasis. These 4 cases can therefore be included among those of acute metastatic gonorrheal dacryoadenitis. The first case of acute dacryoadenitis due to gonorrhea in which the pathogenesis was given as metastasis is the one Pes [13a] reported in 1898. In discussing this case, Sgrosso [25b] and

46. Foster, H. E.: Gonorrheal Tonsillitis, J. A. M. A. **94**:791-792 (March 15) 1930.

47. Gonella: Contributo alla dacryoadenita acuta, Ophth. Klin. **3**:241, 1898.

48. Ferry, M., and Morax, V.: Dacryoadénite bilatérale au cours de l'infection blennorrhagique, Rec. d'opht. **24**:585-587, 1902.

49. Vujtečh, K.: Case of Gonorrheal Urethritis with Bilateral Gonorrheal Dacryoadenitis Due to Infection from Gonococci in Conjunctiva, Časop. lék. česk. **68**:122-124, 1929; abstracted, Zentralbl. f. d. ges. Ophth. **22**:109, 1929.

Sciememi [50] reported 1 case each. Terson [51] in 1900 reported a case of acute metastatic gonorrheal dacryoadenitis. Grimaldi [52] reported 1 case in 1902, Étiévant [22] 2 cases in 1903, Cause [53] 1 case in 1904 and Pes [13b] 3 additional cases in 1904. Casali [54] and Giani [55] reported 1 case each in 1906, Green [56] 1 case in 1907, Wiener [14] 1 case in 1909, Lindahl [57] 2 cases in 1910, Romer [58] 1 case in 1928, Panico [59a] 1 case in 1930, Salvati [10] 7 cases in 1932, de Petri [60] 2 cases in 1932 and Dollfus [61] 1 case in 1938. Wiener [14] mentioned a report of gonorrheal dacryoadenitis by Prioux in 1903, but this I was unable to verify. These 39 cases of gonorrheal dacryoadenitis are published in 26 case reports. They do not include the cases of Sidler-Hugenin, Haab or Prioux. The reports of Pes are outstanding from a clinical and laboratory point of view and for a review of the literature. The paucity of reports from the United States on the subject is remarkable, since the first such report was by an American, Miller.[39b] The reports of Green [56] and Wiener [14] are the only other American reports available. I was unable to find reports on the subject in the British literature. As late as 1923 a discussion of the metastatic gonorrheal lesions of the eye by the Medical Society for the Study of Venereal Disease (London) [62] made no mention of dacryoadenitis. The Italian literature is the most abundant on the subject.

Metastatic dacryoadenitis occurring in gonorrhea differs little in symptoms, course and sequelae from dacryoadenitis from other causes.

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50. Sciememi: Dacrioadeniti nel blenorrhagia, Ann. di ottal. **28**:78, 1898.

51. Terson, A.: Dacrioadénite blennorrhagique, Rec. d'opht. **22**:162-163, 1900.

52. Grimaldi, E.: Contributo alla causistica della dacrioadenite, Gior. internaz. d. sc. med. **25**:344, 1902.

53. Cause, F.: Metastatische Tränendrüsenentzündung bei Gonorrhoe, Ztschr. f. Augenh. **11**:399-404, 1904.

54. Casali, A.: Due casi di dacrioadeniti acuta, Ann. di ottal. **35**:191-206, 1906.

55. Giani, P.: Sopra un caso di dacrioadenite bilaterale durante il decorso di blenorragia ureterale acuta, Riv. ital. di ottal. **2**:188-193, 1906; cited by Byers.[63]

56. Green, J., Jr.: Metastatic Gonorrheal Dacryoadenitis, Tr. Sect. Ophth., A. M. A., 1907, p. 190.

57. Lindahl, C.: Two Cases of Acute Dacryoadenitis, Hygiea **72**:679, 1910.

58. Romer: Dacrioadenite Blenorragia, Tratto di oculista, 1928; cited by Panico.[59a]

59. Panico, E.: (*a*) Dacrioadeniti acute bilaterale nel corso di una blenorragia, Ann. di ottal. e clin. ocul. **58**:59-67, 1930; (*b*) Dacryops intermittente, Boll. d'ocul. **12**:524-525, 1933.

60. de Petri, M.: Dacrioadeniti blenorragia, Lettura oftal. **9**:19, 1932.

61. Dollfus, M. A.: Premier essais de chimiothérapie par les dérivés organiques du soufre des complications oculaires de la blennorragie, Ann. d. mal. vén. **33**:292-296, 1938.

62. Horder, T.: Metastatic Gonorrhea, Medical Society for the Study of Venereal Disease, Lancet **1**:1304-1305, 1923.

The condition occurs more often in males, only 7 of the 39 patients being females. The disease occurs most often in the third decade and a little less often in the fourth. Dacryoadenitis from other causes, in contradistinction, occurs most frequently in childhood and more often in females. The youngest age for gonorrheal dacryoadenitis was 16 years (Key); the oldest, 57 (Pes). Byers [63] observed that the ocular manifestations of systemic gonorrhea were more frequent in young males. The condition is predominantly bilateral, although unilateral occurrences were described by Étiévant,[22] Pes,[13] Sgrosso,[25b] Maklakow,[45] Lindahl [57] and Wiener.[14] The primary lesion is in every instance genital. The lacrimal gland may become involved in the acute stage of the gonorrhea, during an apparently quiet interval or during a recrudesence. In view of the severity of the symptoms, the course is one of surprisingly rapid subsidence, lasting from five days to three weeks and often there are no untoward sequelae. Cases in which the duration was a month were reported by Pes,[13] Maklakow [45] and Terson.[51] The complications of the condition include arthritis, iritis, keratoiritis, glottic edema, inflammation of the salivary gland, metastatic conjunctivitis, lymphadenitis, convulsions, abscess formation and dacryops. Reports suggest that the association of joint involvement is less frequent with gonorrheal dacryoadenitis than with metastatic gonorrheal conjunctivitis.

It is thus evident that acute bilateral metastatic gonorrheal dacryoadenitis is an established clinical entity and is not the medical curiosity suggested in some of the textbooks. Stock [11] in 1925 decided that "The cases in which existing urethral gonorrhea gives rise to dacryoadenitis by means of metastasis are not so very rare." It is true, however, that the condition occurs infrequently, so infrequently in the experience of any one investigator, in fact, that few have been privileged to observe a sufficiently large number of cases for extensive study. Pes [13] with his 4 cases and Salvati [10] with his 7 are unique in this regard. The following single case is therefore considered sufficiently unusual to warrant recording, since it presents some hitherto unreported clinical features and particularly since a histologic study was made of one of the diseased glands. There is only one report on the histopathology of gonorrheal dacryoadenitis in the available literature, that by Morax [63a] in 1902 on a case observed by Ferry and Morax.[48]

II. REPORT OF A CASE

A. CLINICAL COURSE

J. G., a 30 year old Negro, was examined at Provident Hospital on April 7, 1940 because of swelling and redness of both eyes. He had gone to bed on

63. Byers, W. G. M.: Study of the Ocular Manifestations of Systemic Gonorrhea, Stud. Roy. Victoria Hosp., Montreal **2**:1-131, 1908.

63a. Morax, V.: Dacryoadénite blennorhagique, Ann. d'ocul. **128**:448, 1902; Pathologie oculaire, Paris, Félix Alcan, 1929, p. 61.

April 5 with a slight aching and burning of his eyes. The next morning there were swelling and redness of his lids. Home remedies were of no avail, and the eyes began to secrete "pus." There was no headache, only slight photophobia and marked tearing. There was slight malaise but no chills or other general symptoms. He had never had any visual disturbances or worn glasses. For two days before the onset of the ocular symptoms he had suffered burning on urination and frequency and had had a discharge from the urethra. Four years previously he had suffered a "swelling of both eyes" exactly like the present condition but more severe. This was treated by a "specialist" with external medication and cleared slowly in about four to five weeks. This first inflammation as well as the second followed soon after the beginning of a urethral discharge for which the patient had no treatment. There had been no joint pains and no unpleasant sequelae. There had been no other ocular disease. The patient had had no illnesses except the usual diseases of childhood.

The vision was 20/30 in the right eye and 20/25 in the left. Examination of each eye disclosed slight edema of the lids, greatest in the outer thirds, and pseudoptosis. At the external canthus was a chemotic, boggy, pale mass, apparently the lacrimal gland. The left gland had a gelatinous appearance. The right preauricular lymph node was enlarged and tender. The palpebral conjunctiva was diffusely and intensely hyperemic. The vessels in the interpalpebral portion of the bulbar conjunctiva were injected. The bulbar conjunctiva was slightly chemotic. The cornea was clear. There was copious tearing. The tactile tension was normal. There were no cells in the anterior chamber. The slit lamp microscope revealed a finely interlacing, weblike, nonpigmented persistent pupillary membrane stretched across each pupil. Each lens was clear, and there were no lesions in the vitreous or the fundus. The patient had a copious mucopurulent urethral discharge, with edema of the mucosa of the meatus. The tentative diagnosis was gonorrheal conjunctivitis from gonorrheal urethritis by external contamination. Smears were made of material from the conjunctival sac and from the urethra and cultures of the former. Ten per cent neo-silvol solution and cold compresses were ordered. The patient was sent to a urologist for treatment of his urethritis. Abundant gonococci were found in the urethral smear. Only a few diphtheroid bacilli and no gonococci were seen in the eight smears of conjunctival secretion and scrapings. In the cultures of conjunctival material (mediums: blood agar and egg yolk) there were observed diphtheroid bacilli, hemolytic staphylococci and a gram-negative diplococcus, which fermented maltose and was not the gonococcus. The Wassermann reaction of the blood was negative. Three days later the vision was reduced to 20/80 in the right eye and to less than 20/200 in the left. The reduction was due to a wrinkling of the corneal epithelium, which was glassy in appearance but not opaque at any point. This condition seemed to be due to the too vigorous use of cold, for the patient had applied ice cubes directly to the lids. There was an increase in the edema of the lids, and the conjunctiva and the secretion became more mucoid. By this time the increase in size and the inflammation of the lacrimal gland clearly dominated the picture and a diagnosis of dacryoadenitis was made. Drops of atropine sulfate solution and 15 grains (0.97 Gm.) of azosulfamide four times a day were prescribed. An initial injection of 10 cc. of a solution of the latter drug was given. In each eye the cornea became clear and the vision rose to 20/30 in a few days. The chemosis decreased sufficiently to permit detailed examination of the lacrimal gland with the slit lamp. Grossly the left gland was the larger, and its tip appeared cystic (fig. 2). The corneal microscope revealed that the superficial epithelium of the lacrimal gland was gelatinous in appearance and that just beneath it were numerous closely placed

punctate reddish areas which appeared to be either capillary aneurysms or tiny punctate hemorrhages. Interspersed among these were yellow-white spots only slightly larger than the red dots and much less numerous. These were immediately beneath or in the epithelium of the gland and were either clumps of leukocytes or dilated excretory ducts filled with leukocytes. By retroillumination with the slit lamp beam the tip of the left gland was poorly translucent. The actual extrusion of secretion (mucus, pus or lacrimal secretion) by a particular duct could not be made out. Lying deeper were some branching white lines similar to the scarring which occurs in the conjunctiva after chronic inflammation.

Conjunctival scrapings were again examined by smear and the cultures were repeated on the second and the fifth day, and because no gonococci were found the metastatic nature of the condition was suspected. The patient had no symptoms of joint involvement or of cardiac distress. The heart sounds were normal. The only sign of involvement of any other part of the body was a congestion of the nasal mucosa. Examination of the nose showed extensive gelatinous hypertrophy

Fig. 2.—The eyes of J. G. photographed twenty-one days after the appearance of the initial symptoms: (*a*) the cystic tip of the left lacrimal gland, present since the fifth day; (*b*) tag of mucopus.

of the mucosa of the turbinates and septum which seemed to be more than edema. There was a thick tenacious straw-colored mucoid discharge, which formed thick straw-colored crusts, at times producing an almost complete mold of the narrow nasal passages. No bacteriologic studies were made of the nasal secretions. An involvement of the nose on the same side in a case of unilateral dacryoadenitis was reported by Lindahl.[57]

For two weeks there was no appreciable change in the size of the glands. The vision remained normal. The pseudoptosis and the thickness of the lids continued. The secretion diminished in amount, and the subjective symptoms improved. The palpebral conjunctiva remained brick red, with a velvety texture. There was no marked follicular hypertrophy. The bulbar chemosis became localized in the upper temporal quadrant and the upper fornix. The left gland developed a yellowish-tinged cyst about 5 to 6 mm. in diameter. This cyst could not be transilluminated until the third week, when its upper third permitted passage of light and the impression was given of a definite "fluid level," the upper third being milky white

and the lower two thirds yellowish white. Twenty days after the first slit lamp examination there was no evidence of the yellow color and the cyst was uniformly whitish. The conjunctival secretion exuded chiefly from the site of the lacrimal gland, where a tag of thick yellow mucopus would reform after it was wiped away (fig. 2). This pus and scrapings from the conjunctiva were examined by smear. Puncture of the cyst produced about 1 cc. of turbid fluid, which was plated on specially prepared blood agar plates. Cultures also were made of material from the conjunctiva. These were all incubated in carbon dioxide. The laboratory reported that there was a growth of a gram-negative diplococcus which fermented maltose and therefore could not be considered the gonococcus. Because of the slow response of the condition to treatment and because of the otherwise negative laboratory findings it was felt that biopsy would provide some helpful information, and on May 7 a portion of the palpebral lobe of the left lacrimal gland including the macroscopically visible cyst was excised. This procedure is admittedly open to question. Pes [13] questioned its justification when it was employed by Morax [48] because it was "not without danger" and because the acute symptoms of the disease had already passed. Sourdille,[18] however, removed biopsy specimens in 3 cases of dacryoadenitis during the acute stage without ill effect. Since that time numerous investigators have with impunity removed lacrimal gland tissue for biopsy as well as removed the palpebral portion of the gland. In the present case there was no undue reaction to the operation during the still active infective process. The following day there was increased swelling of the left upper lid, but three days later the two upper lids were equal in size though thicker than normal. The cut surface of the left gland healed by granulation. Ten days after the surgical procedure the lower part of the palpebral and the bulbar conjunctiva were no longer injected. There was a contracting scar over the surface of the left gland. Slight secretion remained, and the conjunctiva of the upper lid and of the fornix remained diffusely injected but was not "brick red" as before. On June 21, ten weeks after the condition began, the redness and the secretion had completely disappeared. The lower lids were of normal thickness, but the upper lids were thicker than normal, especially in the temporal halves. The right gland could be seen only when the upper lid was everted, and the left gland was even then barely visible. The vision was 20/25 in the right eye and 20/20 in the left. The palpebral fissures were somewhat narrow, and the patient had the so-called "heavy-lidded" appearance. The dacryoadenitis was considered cured.

On July 2 the patient returned, again complaining of pain and swelling of the eyes. Five days previously he had imbibed alcohol and engaged in sexual excess. Two days later there was burning of the urethra followed by the appearance of a discharge. Three days after the latter he noticed burning of the eyes. The next morning there was swelling of the eyes along with secretion. When the patient was seen there was extreme malaise, with pain about the eyes and head. The vision was 20/60 in the right eye and 20/40 in the left. In each eye there was the typical edema of both lids, greatest in the outer half of the upper lid. There was a thick mucoid discharge from the conjunctiva. The palpebral conjunctiva was diffusely hyperemic, and the upper temporal portion of the bulbar conjunctiva was chemotic. The chemosis caused this part of the conjunctiva to be folded down over the cornea (fig. 3). There was considerable tearing. There was swelling of the lacrimal glands, the left gland being considerably smaller than the right. The condition was clearly a recurrence of the previous dacryoadenitis. The temperature was 101.8 F. and the pulse rate 104. No gonococci were found in the smears of conjunctival material or in the cultures of conjunctival material

and of blood taken at this time. Because of repeatedly negative cultures and smears, a complement fixation test was done; the reaction was reported positive for gonorrhea by the Illinois state public health laboratory. Blood cultures were repeated and were again negative. There was a recurrence of the formation of crusts and the obstruction in the nose. The previous treatment was instituted, but as the progress was slow the azosulfamide was replaced by sulfapyridine (2-[para-aminobenzenesulfonamido]-pyridine). During this recurrence, small discrete opacities appeared on the lower half of each cornea. There were less than ten on each cornea. They were superficial and stained with fluorescein. The epithelium was neither raised nor eroded in these areas. The opacities were not attended by any lowering of visual acuity or by any other symptoms which could be attributed to them. They were observed one week after the appearance of the general symptoms and lasted one week. In twenty-four days the edema of the lids, the chemosis and the mucoid secretion had disappeared, leaving only enlargement

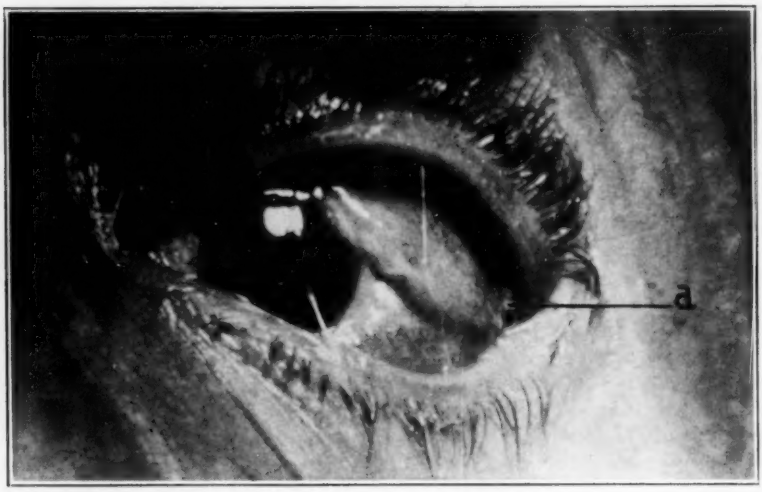

Fig. 3.—Photograph taken four days after the first recurrence, showing the characteristic localized bulbar chemosis and the excessive mucoid secretion: (*a*) the remains of the left gland barely visible in the angle.

and hyperemia of the gland and hyperemia of the upper conjunctival fornix. In the tip of the right gland was a clear cystic area similar to but smaller than the one previously seen in the left. This area did not become as large or as clearly demarcated as that in the left gland.

Almost exactly one month after the first recurrence, on August 3, and before healing was complete, the patient returned with the complaint of swelling of the lids and an increase in all the other symptoms. There had been an increase in the urethral discharge the previous two days. The patient denied sexual contacts or the use of alcohol. About five days before he had received an intramuscular injection from his urologist. There was a severe reaction on the left forearm consisting of a red weal 2½ inches (6 cm.) in diameter, raised about 0.5 cm. Examination of the eyes showed no reduction in vision and only slight edema and redness of the lids, with no chemosis of the conjunctiva and no change in the condition

of the glands from that of the previous examination. The temperature was 99.4 F. and the pulse rate 90. The treatment of the dacryoadenitis was intensified, but two days later the patient returned with an increased swelling of the lids. The characteristic chemosis had become "full blown." There were hyperemia in and secretion from the conjunctiva. The concretions and the obstruction in the nose had returned. The lacrimal glands were only moderately increased in size. Smears and cultures (of material from the conjunctiva and of blood) were again negative for gonococci. Four days later there was less chemosis but the injection, secretion and thickness of the lids remained the same. The concentration of sulfapyridine in the blood at this time was calculated to be 5.12 mg. per hundred cubic centimeters. Two weeks later there was but slight improvement in the condition. The glands were still enlarged and there were still secretion and hyperemia, but the chemosis had disappeared. The patient reported that the urethritis was still active. When he was last seen, thirty-three days after the beginning of the last "attack," the vision was 20/30 in the right eye and 20/25 in the left. The lids had returned to almost normal thickness. The glands were hypertrophic but not discolored, and there was no secretion. The condition was apparently cured.

The tendency to recur is one of the striking features of this particular case. It seems definite, from the patient's description and from the subsequent course, that the "eye trouble" which he had suffered four years previously was an attack of acute gonorrheal dacryoadenitis as severe if not as prolonged as any of the three subsequent attacks. The second attack of dacryoadenitis (the first one described in this report) can therefore be considered a recurrence. It is open to question whether the third attack (the second under observation) was a recurrence or an exacerbation, as it followed the second attack with such rapidity (in less than ten days). However, since between these two attacks there was apparently a clinical cure, it would seem that this represented the second recurrence. The fourth attack was clearly a relapse, an exacerbation or a focal reaction due to the injection of gonococcic vaccine. Byers [63] concluded that the tendency to relapse or recur is one of the features of metastatic gonorrhea. The only other case on record of recurrence of dacryoadenitis due to gonorrhea was that reported by Seeligsohn.[8] The first report of a recurrence of dacryoadenitis from any cause was by Fulton.[64] He treated a young woman for dacryoadenitis who had had the same condition three years previously. A most extraordinary example of recurrence of inflammation of the lacrimal gland was reported by Lagrange and Armaignac.[31] In a woman who had been menstruating normally and regularly since the age of 16 except during four normal pregnancies, there occurred violent dacryoadenitis after the third pregnancy (at the age of 33), which was repeated monthly concurrently with the menses and ceased only after the menopause.

64. Fulton, J. F.: A Case of Acute Primary Inflammation of the Lacrimal Gland Occurring Twice in the Same Individual, Arch. Ophth. **14**:161-163, 1885.

B. PATHOLOGIC CHANGES

Microscopically the section was seen to consist of three lobules of the parenchymatous portion of the gland separated from the conjunctival epithelial surface by a comparatively thick layer of connective tissue showing a marked cellular inflammatory reaction (fig. 4).

1. *Conjunctival Epithelium.*—The conjunctival epithelium varied in thickness from three to eight cells—generally being eight cells thick. The basal layers dipped in to form an actual papilla at one or two points. The basal three

Fig. 4.—Biopsy specimen of left lacrimal gland (× 50): (*a*) conjunctival epithelium; (*b*) submucosa; (*c*) localized edema in the hypertrophied connective tissue stroma; (*d*) parenchyma; (*e*) distended extralobular excretory duct.

to four layers were low cuboidal with large clear nuclei and scant protoplasm. Between the basal two layers were long, spider-like, pigment-bearing cells with processes running in a direction parallel to the surface for a distance of three or four cells. The superficial three to five layers of the epithelium were squamous and pink staining (hematoxylin and eosin), and the most superficial layers were flattened and cornified. Polymorphonuclear cells could be seen infiltrating from the submucosa through the basal layers. In the upper three to five layers were

numerous pus cells, small and large lymphocytes and polymorphonuclear leukocytes, the last-named predominating. A large number of eosinophils was seen. Interspersed among the superficial layers could be noted large goblet cells, some of which opened onto the surface. No bacteria could be found in the epithelium or in the invading pus cells.

2. *Submucosa.*—The submucosa was thickened (two to four times) by the proliferation of fibroblasts and young connective tissue cells, through which were distributed numerous inflammatory cells. Over a considerable portion of the section the submucosal fibers were widely distended by edema. Among the inflammatory cells lymphocytes predominated. There was a remarkable number of eosinophilic polymorphonuclear cells and eosinophilic plasma cells (plasmacytoid). Plasma and some epithelioid cells were also present. No giant cells or caseation was seen. Widely distributed through the submucosa were numerous small, new-formed capillaries, with walls of one cell thickness, large enough to accommodate one to four erythrocytes. Some of the new-formed capillaries were close to the basement membrane of the conjunctival epithelium, indeed were separated by only a one cell layer. There were numerous whorls consisting of three to four concentrically placed endothelial cells, which were considered "precapillaries." These contained no erythrocytes. There were, in addition, an unusually large number of the usual formed capillaries. In some areas the connective tissue just beneath the conjunctival epithelium was infiltrated with collections of red blood cells not in vessels. These may have been due to the trauma at the time of the removal of the tissue, but their distribution suggested that some had occurred before. The inflammatory changes were not uniform all along the submucosa, being particularly marked in certain areas, although not to the degree of abscess formation in any one area.

3. *Supporting Tissue.*—The supporting connective tissue was much proliferated and filled the bulk of the section (fig. 4). Traversing the connective tissue were the larger blood vessels and a number of small and large epithelial-lined channels, the extralobular excretory ducts. Except around the ducts, the inflammatory reaction in the connective tissue was scant when compared with that in the submucosa. These ducts were seen in both longitudinal and cross sections. One series of sections showed not only the large cystic space seen clinically, which proved to be a widely dilated primary excretory duct, but the point of emergence of this duct on the conjunctival surface.

4. *Excretory Ducts.*—(*a*) Extralobular Ducts: Three types of extralobular ducts could be made out:

(1) Contributory: Small contributory ducts had an epithelial lining two to three cells thick, of the low cuboidal type, and had open lumens. The epithelium was arranged in an orderly manner, and the space around the ducts was comparatively free from inflammatory reaction except where the ducts emptied into the larger ducts.

(2) Primary: The second type included the ducts of the size described in the structure of the normal gland which open on the conjunctival surface. Around these ducts there was observed a characteristic type of inflammatory reaction. A dense "collar" of pus cells surrounded each duct; pus cells permeated the epithelium and were present within the lumen. Under the higher magnifications the reaction was seen to consist of a collection of fibroblasts, plasma cells, small and large lymphocytes and polymorphonuclear leukocytes. The fibroblasts, plasma cells and lymphocytes were more numerous in the periphery of the collection. More centrally, in the widely distended spaces of the lamina propria of the duct epithelium, the poly-

morphonuclear leukocytes predominated. Many of the polymorphonuclear leukocytes in this location as well as in the lumen of the duct were undergoing degenerative changes, such as fragmentation and fatty degeneration. The duct epithelium was proliferated to six to eight layers, was infiltrated by pus cells, was of the low cuboidal variety and showed considerable toxic damage. The cytoplasm of the epithelial cells was edematous, and the nuclei in some instances showed marked fatty degeneration, pyknosis and karyolysis. There was proliferation of goblet cells in the duct epithelium, in places even to the point of "mucoid degen-

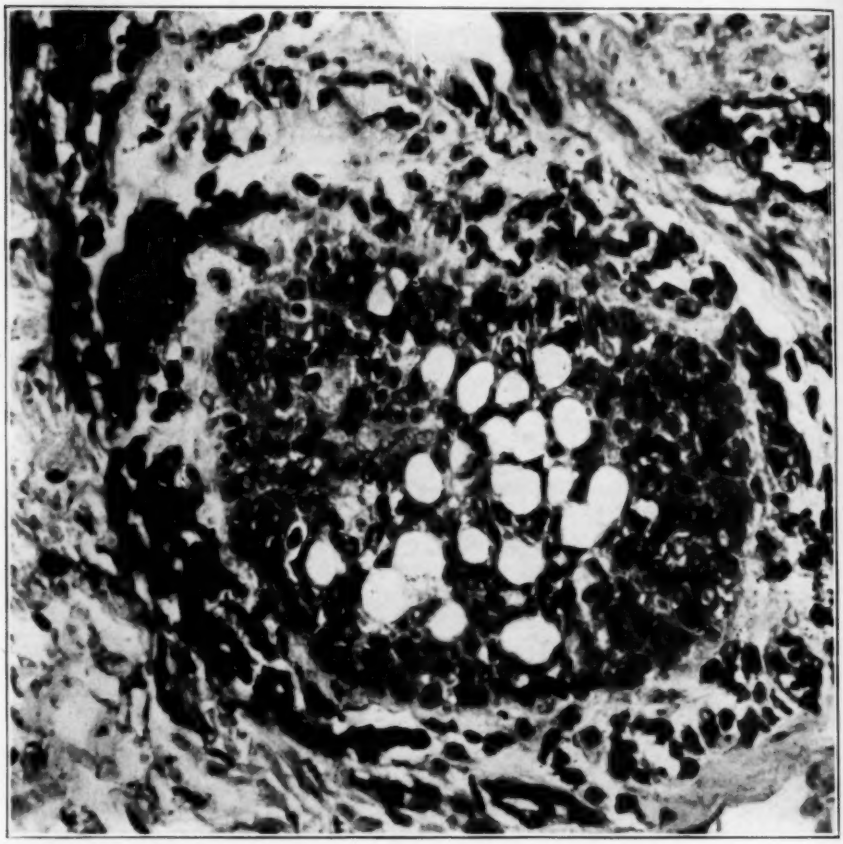

Fig. 5.—Extralobular excretory duct (× 450), showing occlusion by epithelial proliferation, distention of "goblet cells" and infiltration of the lamina propria by inflammatory cells.

eration." Within the lumen was a collection of degenerated epithelial cells, pus cells and mucus. This combination of toxic inflammatory reaction, epithelial proliferation, mucoid degeneration and debris served to diminish greatly the lumens of the ducts, and in one section a duct was seen to be completely occluded by the process (fig. 5). When successive sections were examined it could be seen that as the ducts approached the conjunctival surface the cellular debris filling the lumens became more amorphous.

(3) Dilated: The third type consisted of ducts widely dilated beyond the normal, apparently by blockage (fig. 6). The size varied from twice the normal to that of the large cystic space seen clinically. The epithelium was only from two to four layers thick, the inner layer being cuboidal and the outer layers

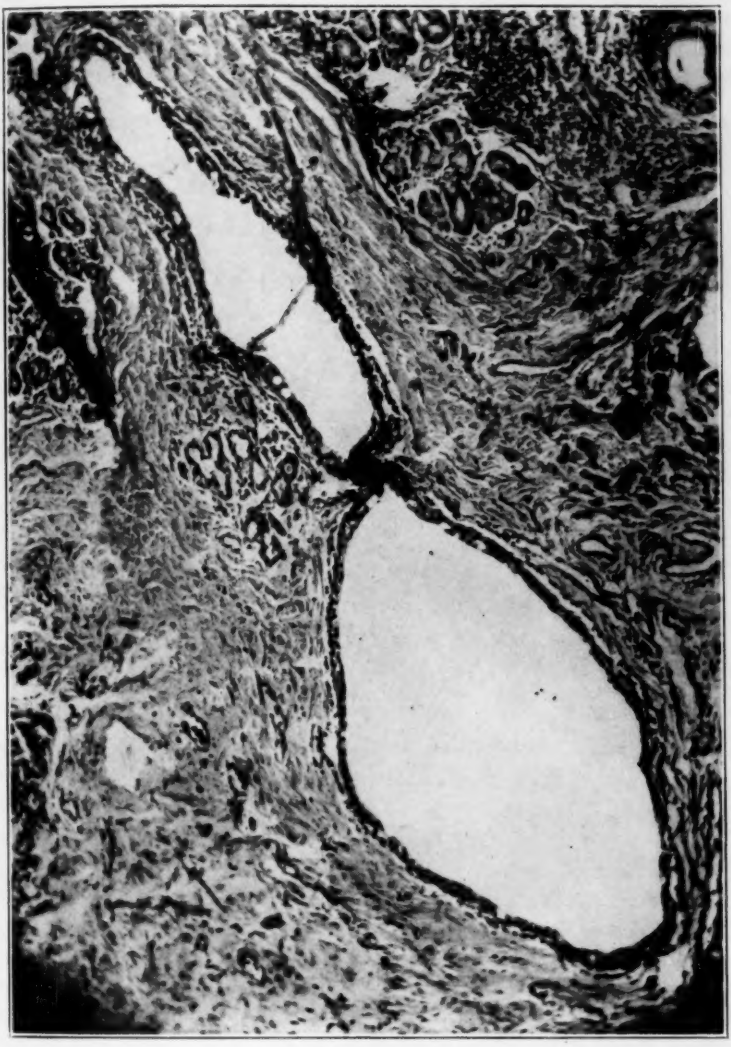

Fig. 6.—Two separate extralobular excretory ducts just after bifurcation (× 98), showing moderate distention.

flattened to a squamous character. There was some edema of the cells, but none of the marked degenerative changes seen above. There were a few pus cells infiltrating the epithelium, but the collar of inflammatory cells was absent or greatly reduced. Goblet cells were present but fewer than in the conjunctival epithelium

and at places seemed to extend the whole thickness of the lining of the duct. The cyst seen macroscopically was found to be one such duct in a state of extreme dilatation, an actual dacryops. Through various sections this duct could be seen to open onto the conjunctival surface by means of a short thick channel which led from a large epithelial-lined cavity (fig. 7). The inflammatory reaction around the mouth of this duct differed little in type or in degree from that found under the conjunctival epithelium, with this notable exception: In the angle formed by the mouth of the duct and the conjunctiva was a large, ovoid, compact focus

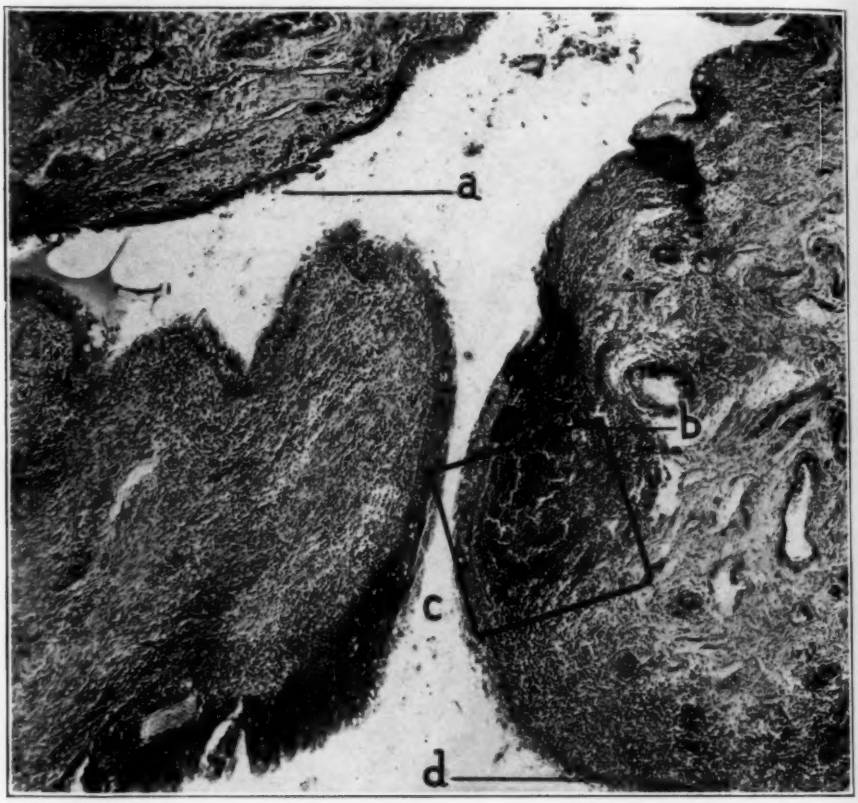

Fig. 7.—Opening of the dacryops onto the conjunctival surface (× 65): (*a*) cavity and walls of the dacryops; (*b*) lymph follicle (the rectangle is the area shown in figure 8); (*c*) mouth of the duct opening on (*d*) the conjunctival surface. The width of the duct (*c*) was somewhat exaggerated in mounting. The plug of mucus in the mouth of the duct referred to in the text does not appear in this section.

of lymphocytes and connective tissue reticulum. The cells were arranged in a roughly concentric fashion and were surrounded by an ill defined connective tissue "capsule," beyond which were the usual inflammatory cells (fig. 8). Anatomically, at least, this was an actual lymph follicle, and it was located just beneath the lamina propria of the duct epithelium. At its mouth the duct epithelium was six to eight nuclei thick and showed considerable mucoid degeneration. The lumen was filled by a plug of mucus. In the walls of the cyst the epithelium

varied from squamous to cuboidal in type and was from one to four layers in thickness. There were areas in which the epithelium was up to eight layers thick despite the apparently great amount of "distention." The contents of the cyst were an albuminoid amorphous substance in which there were a few erythrocytes and degenerated pus cells. The folds in the walls of the cyst were such as might have resulted from its collapse after aspiration.

(*b*) Intralobular Ducts: The intralobular ducts varied from equal in size to twice the size of the alveoli. The epithelium was one to three layers—generally

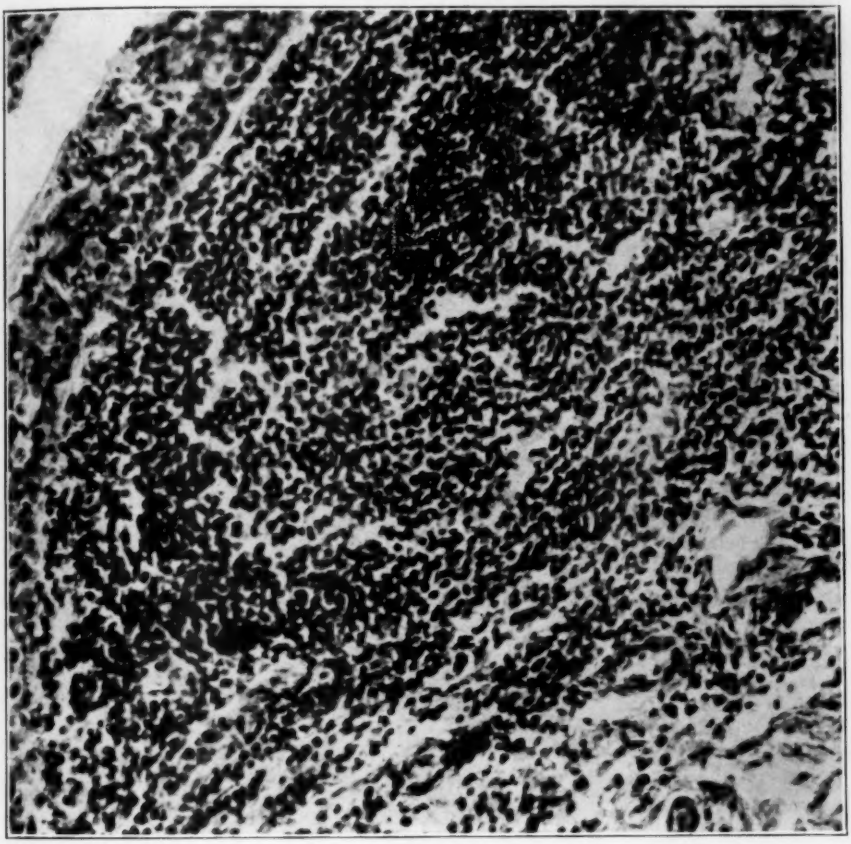

Fig. 8.—Detail of the lymph follicle shown in figure 7 (× 310).

two layers—thick. It was of the low cuboidal type. The cytoplasm showed edema, not severe but generalized and giving it a fine, loosely reticular appearance. There was no fatty degeneration, karyolysis, etc. An occasional pus cell could be seen permeating the epithelium but no goblet cells and no mucoid degeneration. Surrounding the intralobular ducts there was a collection of lymphocytes such as is found between the alveoli of the parenchyma. The intralobular ducts gave the impression of having participated in a toxic damage of the gland but not in the cellular inflammatory reaction.

5. *Parenchyma.*—The secretory portion of the gland showed little gross inflammatory change. The interalveolar connective tissue was infiltrated with lymph

and plasma cells, which were clumped in certain areas but not to the degree of actual follicle formation. There were also young fibroblasts and large mononuclear cells in the interalveolar connective tissue, with cytoplasm containing large deep-staining particulate matter, resembling cellular debris and suggesting phagocytosis. The nuclei of some of these cells showed, also, multiple vacuoles. Plasma cells and plasmacytoid cells were seen in small numbers. No polymorphonuclear leukocytes were seen in this area. The secretory epithelium showed numerous fat droplets, and in addition the cytoplasm contained a large number of

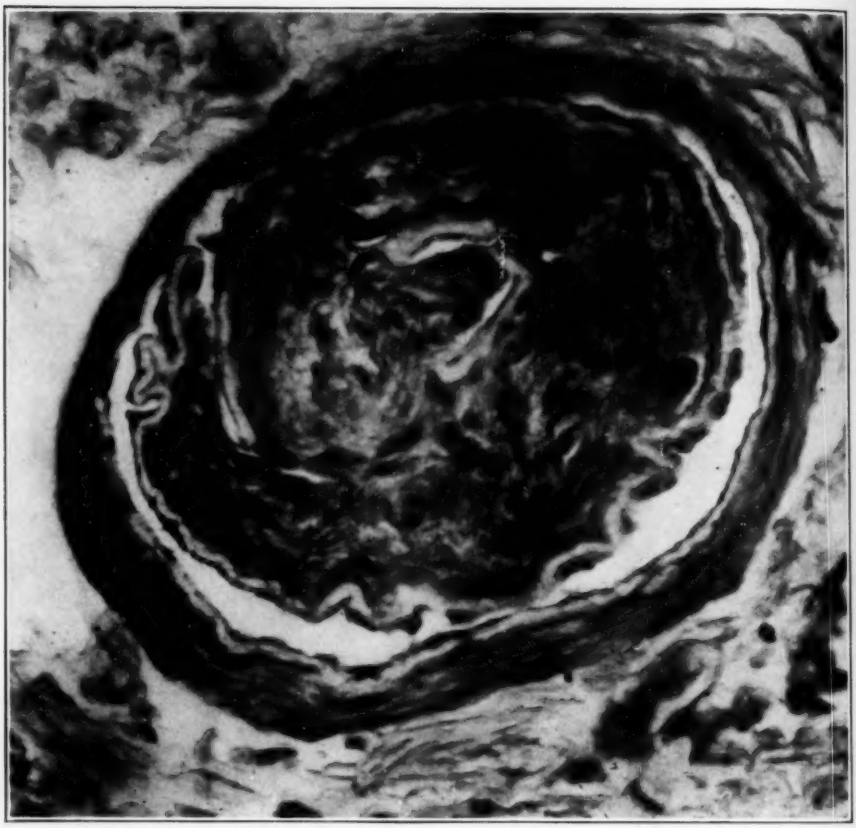

Fig. 9.—Thrombotic blood vessel (× 750), showing organization and recanalization. The slitlike space in the hyalinized media is an artefact produced in mounting.

droplets, probably secretory, giving the impression of hydropic degeneration because of the swollen, pale-staining appearance of the cells. The detailed observations indicate that the parenchyma of the gland was actually more involved in the inflammatory process than was at first evident.

6. *Blood Vessels.*—Mention has already been made of the new blood vessel formation in the submucosa. There were, deep in the connective tissue stroma, close to the lobules of the parenchyma of the gland, larger vessels which showed inflammatory changes, such as obliterative arteritis and phlebitis. These changes

involved mainly the intimal and the medial layers. There were necrotic changes in the cells of the media and proliferative and degenerative changes in the intima. In certain vessels these changes had led to the formation of thrombi, with organization and recanalization in a few instances (fig. 9). In one fortunate section, such a vessel had been cut longitudinally for a considerable distance of its length and showed a recanalized thrombus with its endothelial-lined channels. In any one section, recent as well as old, changes could be found, from early phlebitis and arteritis and early thrombus formation to complete organization and canalization. No such vessels were found in the parenchyma of the gland. Perivascular "round

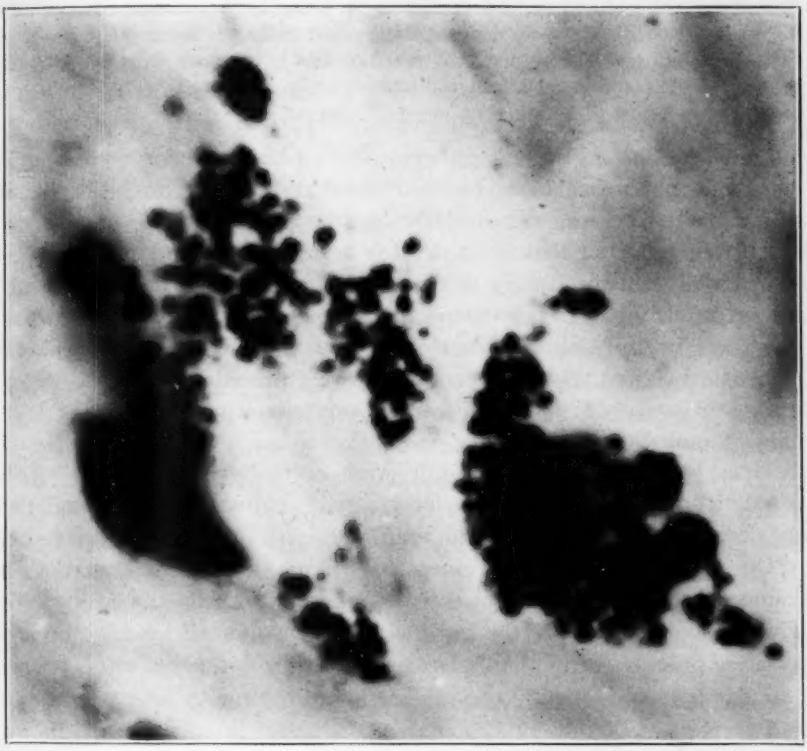

Fig. 10.—Tissue histiocyte (× 3,250), showing phagocytosed coccoid particles. Some of the particles appear to be extracellular and in tissue spaces.

cell" infiltration was found only in the submucosa, where the lymphocytic infiltration was thickest.

7. Bacteriologic Observations.—In the immediate vicinity of the larger vessels, both arterioles and venules, were large mononuclear tissue cells with a generous cytoplasm. The nuclei were about one third the size of the cell and in some instances were slightly indented. The cells varied from spindle to ovoid in shape. These cells were not found in the conjunctival epithelium or in the parenchyma. The number varied from about two to six around a particular vessel. The cytoplasm of these cells was filled with numerous large, deep-staining "granules" (fig. 10). The majority of the "granules" in any one cell were uniform in size

and coccoid in shape. In some of the cells these were closely packed and were the only type of inclusion to be found. In quite a few of the cells, however, the inclusions were less densely packed and in addition there was larger, coarser, irregularly shaped particulate matter. Some of the "granules" were seen in tissue spaces, apparently extracellular. With the Giemsa stain for bacteria in tissue, these "granules" were stained a deep blue color; with the Masson tetrachrome stain,[65] the inclusions were stained by the orange G stain; when the sections were stained for gram-negative organisms in tissue, the coccoid particles took the dark red stain, while the larger particles were stained blue. Morphologically the cells were tissue mast cells, but because of the wide variation in the size of the cells and in the size and the distribution of the cytoplasmic inclusions and because of the extreme difficulty in demonstrating mast cells in human tissue without special fixation and staining (which were not used), the possibility that some of these inclusions represented bacteria was strongly suggested. The significance of such a concept will be taken up under "Comment."

8. *Comment.*—The only other study on the pathologic changes of metastatic gonorrheal dacryoadenitis is that reported by Morax,[63a] in which the tissue was removed during the subacute stage of the disease. He reported that certain lobes were normal and others represented well defined changes in the secretory epithelium which consisted in the infiltration of polymorphonuclear leukocytes. There were no gonococci in the tissues. A second portion of tissue was incubated in ascitic boullion, but no gonococci were cultured. Of the histologic studies of acute dacryoadenitis, the observations in the ones by Sourdille [18a] and Sgrosso [25a] agree most closely with the observations in this case. The cases of Sourdille were of unilateral abscesses of the gland, from which only staphylococci were cultured. He stated that the lesions were both canalicular and pericanalicular. The epithelium of the ducts was swollen, proliferated and cast off in the lumen. The connective tissue was infiltrated with "embryonal" cells and round cells. The secretory portion of the lacrimal gland was not affected by the suppurative process but by the "sclerosis" of the tissues. Sgrosso described vascular lesions consisting of proliferation of the tunica interna, swelling and necrosis of the fibromuscular elements and obliteration of the lumen. He called the condition the degeneration of Löwenfeld.

The pathologic changes in the case reported represent tissue changes present after thirty days of the disease and must be interpreted in that light. In reconstructing the pathologic process on the basis of the findings it must be concluded that the far advanced vascular changes (such as organized thrombi and canalization) indicate that the vessels

65. Masson, P.: Trichrome Stainings and Their Preliminary Technique, J. Tech. Methods **12**:75-90, 1929. Goldner, J.: Modification of Masson's Trichrome Technique for Routine Laboratory Purposes, Am. J. Path. **14**:237-243 (March) 1938.

became involved early in the process and likely constituted the initial lesion of the lacrimal glands. The canalization may even represent a residue from the first attack, four years ago. These changes in the blood vessels are especially significant in light of the experience of Radnot,[43] who found no evidence of thrombosis in 500 diseased lacrimal glands examined. Production of thrombi is characteristic of the disturbed physiology caused by the gonococcus (Jadassohn,[66] p. 75). The pathologic changes of acute metastatic gonorrheal dacryoadenitis can therefore be interpreted in the light of (1) the effects on the tissues of the lowered nutrition resulting from the disturbance of the blood supply and (2) the reaction of the tissues to either local bacteria and/or bacterial toxins. The hypertrophic changes in the epithelium as well as the metaplasia (the change from a cuboidal to a squamous form or the reverse) constitute a characteristic gonorrheal pathologic process. The mucoid degeneration of the epithelium of the ducts is both a metaphasia in response to toxins and a hyperplasia of the mucous glands normally present in the lacrimal gland. The presence of a large number of fibroblasts, plasma cells and eosinophils represents the cellular response usually found after the third week of an acute gonorrheal process, as pointed out by notable authorities on this subject, Bumm, Thayer, Wertheim and Jadassohn.[66]

Sselkow,[68] after extensive study of the gonorrheal exudate, concluded that histiocytes, which arise from fibroblasts, immobilize the gonococci by adsorption and later agglutinate them into a so-called resorption body, which is gram-positive in its staining and considerably larger than the gonococcus. During this process the endotoxin is liberated and is absorbed by the red blood cells, this reaction being accompanied by a febrile reaction. A later phase of this continued process is the formation of eosinophils from the histiocytic cells. The whole process, from the beginning of the disease to the formation of eosinophils, takes about seventeen days. The atypical "mast cells," with their various-sized inclusions, observed in the present case, may conceivably represent the phagocytosis of bacteria by histiocytes. Finger, Ghon and Schlagenhaufer [69] concluded that gonococci are prone to be deposited in tissue in slits or spaces rather than diffusely through the tissue.

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66. Jadassohn, J.: Allgemeine Ätiologie, Pathologie und bacteriologische Diagnose der Gonorrhoe, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1934, vol. 20, pt. 1, pp. 1-285.

67. Footnote deleted.

68. Sselkow, E. A.: Die Beteiligung des retikulo-endothelialen Systems an dem akuten Gonorrhöeprozess, Ztschr. f. Urol. **25**:48-60, 1931.

69. Finger, E.; Ghon, A., and Schlagenhaufer, F.: Ein weiter Beitrag zur Biologie des Gonococcus und zur pathologischen Anatomie des gonorrhoischen Process, Arch. f. Dermat. u. Syph. **33**:141-182 and 332-339, 1895.

The preponderance of lymphocytes and leukocytes of lymphocytic origin in the cellular reaction in the submucosa is an expression of the duration of the process. It is likely that polymorphonuclear leukocytes were in excess in the earlier stages of the disease. The presence of the large number of new-formed vessels in the submucosa shows a reparative process resembling the formation of granulation tissue. The relatively greater thickness of the connective tissue over that of the parenchyma is a residue in part from the previous infection. The fact that the secretory portion of the gland is as little involved as it was is in keeping with the accepted concept that the gonorrheal process is primarily an interstitial infection and that glandular structures are involved only secondarily (Finger, Ghon and Schlagenhaufer [69]). The fatty degeneration in the epithelium of the ducts, the edema and the hydropic changes in the parenchyma are all expressions of a severe intoxication. Fat droplets in the parenchyma are a normal finding (Axenfeld [34b]). After reviewing the opinions of Axenfeld,[34b] Arlt,[70] de Schweinitz,[71] Radnot [43] and others, I am inclined to feel that, while the lymphocytic accumulations in the parenchyma and elsewhere may not be "normal," as some investigators claim, they are not necessarily caused by the inflammatory reaction but represent some degenerative process which is an expression of age. It is to be expected, however, that they would have participated in the inflammatory reaction just as did every other part of the tissue. The formation of follicles, as at the mouth of the cyst, is an expression of the extent of their participation. Arlt [70] stated that, along with Waldeyer, he had never seen actual follicle formation in the lacrimal gland. Although numerous pyknotic epithelial cells were seen, there were no cells which could be identified as the oncocytes of Hamperl.[72] The propensity of the cellular exudate in gonorrhea to escape to the epithelial surfaces accounts for the cellular reaction about the excretory ducts.

As to the pathogenesis of the large cystlike dilatation of one of the ducts: It was the opinion of Lange [73] that such dilatations result from blockage of the ducts by debris and scarring, the distention being caused by continued secretion. It is known, however, that blockage of the duct

70. Arlt, A.: Original Contribution Concerning the Glandular Structure Appertaining to the Human Eye and Its Appendages, St. Louis, Beaumont Hospital and Medical School, 1900.

71. de Schweinitz, G.: Anatomy of the Lacrimal Gland, Tr. Am. Ophth. Soc. **36**:182-186, 1900.

72. Böck, J., and Schlagenhauff, K.: Ueber das Vorkommen von Onkozyten in der menschlichen Tränendrüse, Ztschr. f. Augenh. **94**:244-252, 1938.

73. Lange, O.: Zur Anatomie und Pathogenese des Dacryops, Arch. f. Ophth. **47**:503-507, 1899.

of the lacrimal gland is more likely to result in the cessation of secretion from the portion of the gland which the duct drains, such as occurs when the palpebral portion of the gland is removed surgically. In the present case no such dilatation occurred after the biopsy of the left gland. Goldzieher [74] stated the belief that dacryops is an expression of a mucoid degeneration of the gland. In the present case the mucoid degeneration as well as the dacryops was confined to the ducts. There have been cases of dacryops described in which no "goblet" or mucoid cells were seen. Sourdille [18a] expressed the opinion that the condition is due to inflammatory changes about the mouth of the ducts as a result of infection of the conjunctiva and to sclerosis of the tissues about the ducts along with increased secretion of the gland. In the present case there was no evidence of any such organic closure, as mucus could be seen exuding from the mouth of the duct in the microscopic section. Further, the amount of pressure necessary to overcome such organic closure would be enough to cause stoppage of the secretion by pressure "atrophy." Two other factors may be considered as significant with regard to the flow of secretion in the ducts. One of these, the accumulation of lymphoid cells located about the mouth of the larger ducts (fig. 7), has an analogous arrangement in the anatomy of the mouth of the eustachian tube. The hypertrophy of the lymphoid elements in this situation is capable of blockage of the tube. The other factor is that the larger ducts open on the conjunctival surface in an oblique direction, an arrangement which allows for a valvelike action (Arlt [70]). From experimental and anatomic evidence, it does not seem likely that the lacrimal gland possesses sufficient secretory pressure to produce by pressure alone such distention as is found in a dacryops. In view of the low secretory pressure, it is not necessary to postulate any elaborate causes for blockage of a lacrimal duct, and any of the causes mentioned might easily create an embarrassment of the flow of secretion. How, then, is one to explain the distention found in the dacryops? Beattie and McDonald [75] and other workers have shown that there is a nervous as well as a chemical influence on the contractibility of the excretory ducts. Solution of posterior pituitary, for instance, causes contraction of the muscular elements about the ducts expressing secretion. Could not some interference with this chemical or nervous control allow for a more or less passive dilatation of the ducts, or if not a passive dilatation

74. Goldzieher, M.: Ueber die Cyste der Tränendrüse, Dacryops, Arch. f. Ophth. **61**:339-351, 1905.

75. Beattie, J., and McDonald, P. R.: Cytological Changes in the Lacrymal Gland Following the Administration of Certain Drugs, Proc. Roy. Soc., London, s.B **113**:217, 1933.

at least a dilatation by relatively low pressures? I believe that the action of certain toxins along with the cellular infiltration of the walls of the excretory ducts destroys the contractile mechanism in these ducts. When this happens and there is any interference at the mouth of the duct by any of the causes mentioned (and this interference need not necessarily be total) the low secretory pressure of the lacrimal gland is sufficient to cause a distention of the walls of the duct. Further evidence that blockage is not the prime factor in the production of dacryops is seen in the case reported by Panico,[59b] who observed that a dacryops would form in a 36 year old woman after weeping and would disappear after fifteen to twenty minutes. The entire process may be abetted early in its course by the accumulation in the lumen of large collections of inflammatory cells (pseudoabscess formation), by formation of mucus and by increase in the secretory rate and later by the effect of gravity. The fact that some of the ducts are located immediately under the conjunctiva for some extent allows them to be distended to large proportions. The concept of a low pressure in the cysts is supported by the fact that in quite a few anatomic descriptions of dacryops there were mentioned some areas in which the lining of the cyst not only retained its normal cuboidal structure but in some instances even proliferated instead of becoming flattened, as would be expected in an environment of continued high pressure. Folds such as were found in the present case have also been reported in the walls of these cysts. Further support of the view that dacryops is due to some generally operating factors, and not to some purely local (at the mouth of the ducts) causes, as has hitherto been maintained, is had in a recent case in which examination was performed with the slit lamp microscope. The palpebral portion of both lacrimal glands as well as the conjunctival glands presented multiple small dacryops, varying in size from that of a pinhead to that of a bird shot. In the dependent portion of some of these was a yellowish white refractile accumulation, having a sort of "fluid level," consisting of cells, mucus or both. The condition represented the so-called cystic degeneration of the lacrimal gland. Radnot [43] has described such cases microscopically.

In correlating the histologic observations with the picture seen under the corneal microscope, it can be seen that the gelatinous appearance of the conjunctival epithelium of the gland was due to the combination of hypertrophy, edema, leukocytic infiltration and preponderance of goblet or mucoid cells. The punctate red spots just beneath the epithelium were largely dilated and proliferated capillaries, but some were due to subepithelial sheetlike hemorrhages. The yellow-white, subepithelial spots were the follicle-like accumulations of lymphocytes and

superficially placed ducts distended with inflammatory exudate. The deeper-lying white branching lines were excretory ducts with their collars of inflammatory cells, rather than scars. The large cystic space seen grossly was without doubt the dilated excretory duct, which microscopically proved to be a dacryops formation. Its early yellowish color was due to the accumulation of pus cells within its lumen. It was because of this accumulation that the cyst was so poorly translucent in the beginning. The later clearing and increase in translucency were due to slow extrusion of the cells through the partly blocked mouth of the duct, to liquefaction and to dilution of the contents by lacrimal secretion and mucus.

III. COMMENT

According to medical historians (Hirsch [1]), the disease known today as gonorrheal ophthalmia was known to the early Greek and Arabian writers and was ascribed to a variety of causes. It was not until the early part of the eighteenth century that Carl de St. Yves [76] referred to the venereal nature of the condition. He stated that the condition arose because of stoppage of the urethral discharge, which caused "metastasis" to the eyes in order to "liberate the matter." In 1736 Astruc [77] made the first claim on record that the eye could be involved in disease of the genitalia by an exogenous route, by means of the urine. It was Piringer [78] who, in 1841, first gave experimental proof of the involvement of the eyes by gonorrhea, and for some years thereafter the pendulum of opinion swung in the direction of exogenous involvement. At the extreme were Arlt [7] and Ruete,[79] who claimed that all gonorrhea of the eyes was exogenous. Not long thereafter, Fournier [80] described conjunctivitis in patients with gonorrhea in whom he felt that the ocular condition was endogenous. Since then inflammation of the eyes by means of metastasis has become an accepted concept which has had considerable study and proof, both in the clinic and in the laboratory. Metastatic lesions of the eye in the course of generalized conditions are found in association with rheumatic fever, pneumonia, dysentery, typhoid fever, dengue, meningococcic meningitis, staphylococcic and strepto-

76. de St. Yves, C.: Tractat von denen Krankheiten der Augen, aus dem Französischen ins Deutsche übersetzt von J. A. Mischel, Berlin, J. A. Rüdiger, 1730.

77. Astruc, J.: Die morbis venereis libri sez, Paris, G. Cavelier, 1736, vol. 24, p. 192.

78. Piringer, J. F.: Die Blennorhoe am Menschenauge, Grätz, F. Ferstl, 1841.

79. Ruete, C. G. T.: Lehrbuch der Ophthalmologie, Brunswick, F. Vieweg u. Sohn, 1854.

80. Fournier, A.: Ophthalmie blennorrhagique rheumatismale, in Jaccoud, S.: Nouveau dictionnaire de médecine et chirurgie pratique, Paris, J. B. Baillière, 1866, vol. 5, pp. 245-251.

coccic septicemia, tuberculosis and syphilis as well as gonorrhea. Haab [23b] (1881), Axenfeld [34a] (1899) and van Moll [81] (1899) were among the first ophthalmologists to concern themselves with the problem of gonorrheal metastasis to the eyes, a subject which was discussed extensively at the Ophthalmological Congress at Utrecht in 1899. Notable contributions on the subject which have become more or less classic are the studies by Greeff [82] (1905), Beyers [63] (1908), Heerfordt [83] (1909) and Elschnig [26b] (1912). One of the latest and most inclusive reviews on metastatic gonorrhea of the eye is by Fehr.[83a]

It is now established that gonorrhea may involve almost all portions of the eye by metastasis. Mention has already been made of metastatic gonorrheal conjunctivitis. Notable reports on this subject in addition to those already mentioned have been made by Sidler-Hugenin,[23a] Carroll [84] and McKee.[85] Metastatic gonorrheal iritis has been reported by Förster [86] and Breiger;[87] metastatic gonorrheal involvement of the choroid, by Schaeffer-Buchardt;[23c] metastatic gonorrheal keratitis, by Pincus [88] and Martin-Haltenhoff;[89] phlyctenule formation due to metastatic gonorrhea, by Heerfordt [83] and Schieck;[90] thrombosis of the retinal vessels in metastatic gonorrhea, by Galezowski,[91] and papil-

81. Van Moll, F. D. A. C.: Gibt es eine metastatiche Conjunctivitis? Ber. ü. d. Verhandl. d. IX. Internat. Ophth. Kong., Utrecht, 1899; Ztschr. f. Augenh. (supp.) **2**:60, 1899.

82. Greeff, R.: Ueber Metastasen im Auge bei Gonorrhoe, Charité-Ann. **26**: 620-643, 1905.

83. Heerfordt, C. F.: Ueber "Subconjunctivitis epibulbaris gonorrhoica," Arch. f. Ophth. **72**:344-366, 1909.

83a. Fehr, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1934, vol. 20, pt. 2, p. 379.

84. Carroll, J.: Metastatic Conjunctivitis in Gonorrhea, Tr. Sect. Ophth., A. M. A., 1907, pp. 171-192.

85. McKee, S. H.: Metastatic Gonorrheal Conjunctivitis, Ophthalmoscope **5**: 618-621, 1909.

86. Förster: Beziehung der Krankheiten der Harnorgane zum Sehorgane, in Graefe, A., and Saemisch, E. T.: Handbuch der gesammten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1877, chap. 13, pp. 86-87.

87. Breiger, F.: Metastatische Augenkrankheiten bei Gonorrhoe, Dermat. Wchnschr. **83**:1632-1643, 1926.

88. Pincus, F.: Metastatic Conjunctivitis Involving the Cornea, Arch. Ophth. **43**:136-145, 1914.

89. Martin-Haltenhoff, H.: Recherches anatomico-pathologiques sur les inflammations métastiques à la suite de la gonorrhée, Rev. méd. de la Suisse Rom. **2**:308-352, 1882.

90. Schieck, F.: Die Phlyktane als Immunitäts-Erscheinungen, München. med. Wchnschr. **79**:6-9, 1932.

91. Galezowski: Sur les altérations vasculaires de la rétine dans l'infection blénorrhagique, Ann. d'ocul. **123**:301, 1900.

litis due to metastatic gonorrhea, by Schindler.[92] Extrabulbar involvement of the eye by metastatic gonorrhea other than dacryoadenitis includes abscess of the lid, reported by Haab [23b] and Sidler-Hugenin.[23a]

Except for the proximity of the lacrimal gland to the eye and the common source of blood supply there is little justification for considering involvement of the lacrimal gland under the heading of diseases of the eye. A closer homology can be found in the involvements of other glandular structures, such as the salivary and the endocrine glands. Examples of simultaneous involvement of the parotid and the lacrimal glands are seen in Mikulicz' disease and in epidemic parotitis. Curtis [93] in 1887 reported the first case of parotitis in the course of gonorrhea. There have been subsequent reports of metastatic gonorrheal parotitis by Colombini,[94] Lemierre and Faure-Beaulieu,[95] Roques [96] and Steiner and Walton.[97] Colombini was able to recover the gonoccocus from the parotid gland by culture. Attempts at culture by Roques were unsuccessful. Metastatic gonorrheal mastitis was reported by Oxley,[98] who was able to recover the gonococcus from the gland by culture. Metastatic gonorrheal thyroiditis was reported by Alexandresco-Dersca and Jonesco,[99] who were also able to culture the gonococcus from material from the gland. The submaxillary gland was involved in a case of metastatic gonorrheal dacryoadenitis reported by Pes.[13] In the case reported by de Lapersonne [38] the parotid glands and the mammae were involved. Except in the cases of Pes and de Lapersonne, the glandular involvements were all attended by suppuration. Only in the case of Étiévant [22] were gonococci found in the pus from an inflamed lacrimal gland. From these few reports it would seem that metastatic gonorrhea in other glandular structures is considerably more rare than that in the lacri-

92. Schindler, E.: Stauungspapille bei metastatische gonorrhoische Augenkrankheiten, Dermat. Wchnschr. **87**:1297-1299, 1928.

93. Curtis, B. F.: Parotitis Complicating Gonorrhea, New York M. J. **45**:346, 1887.

94. Colombini, P.: Bakteriologische und experimentelle Untersuchungen über einen merkwürdigen Fall von allgemeiner gonorrhoische Infektion, Centralbl. f. Bakt. (Abt. 1) **24**:955-963, 1898.

95. Lemierre, A., and Faure-Beaulieu, M.: Septicémie et pyohémie gonococcique, Gaz. d. hôp. **79**:231-235, 1906.

96. Roques, E.: Parotidites blennorrhagiques, Ann. de dermat. et syph. **1**:94-97, 1910.

97. Steiner, W. R., and Walton, L. L.: Gonorrheal Endocarditis with Bilateral Parotitis and Toxic Jaundice as Added Complications, Arch. Int. Med. **11**:1464-1471 (Feb.) 1938.

98. Oxley, W. H. F.: Gonorrheal Mastitis, Brit. M. J. **2**:744, 1920.

99. Alexandresco-Dersca, C., and Jonesco, D.: Thyroïdite suppurée gonococcique à la suite d'une gonococcémie, Paris méd. **1**:188-189, 1932.

mal gland. There are no reports on the histopathologic picture of gonorrheal inflammation in the other glands, so that a comparison must be limited to clinical and to other laboratory findings in considering any similarity in the pathogenesis of the two conditions.

In the majority of the reports in which the dacryoadenitis has been considered to be metastatic, the author has expressed the opinion that the gonotoxin and not the organism was the causative agent because of the almost uniform absence of positive bacteriologic findings. Salvati [10] did punctures of the gland and took material for culture in all 7 cases which he reported, and the cultures were all negative. For that reason he felt that the condition was toxic in origin. In the light of this observation the finding by Étiévant [22] is doubly significant.

Before one draws any conclusions as to the pathogenesis of metastatic gonorrheal dacryoadenitis, it is logical to consider first not only the basic and accepted aspects but some of the more recent concepts of the process of gonorrheal metastasis itself, including: (1) those circumstances at the site of the primary lesion which favor metastatic spread of the inflammatory process, (2) the channels through which this spread takes place, (3) the specific etiologic agent responsible for the distant inflammatory process, (4) those factors which favor the localization of the etiologic agent at a particular site and (5) those factors which contribute to the activation and reactivation of the agent at the new site.

As to the first of these, the local factors: The overwhelming preponderance of metastatic gonorrheal lesions of many different types, including dacryoadenitis, in males would strongly suggest that there is something in the anatomy or the physiology of the male genitalia which contributes to metastasis. The emphasis which Zentmayer [100] and Thomasson [101] placed on the significance of the prostatic portion of the urethra and the prostate in metastatic gonorrheal ocular diseases is in support of this probability. Fanz and McCrea,[102] after examining a number of postmortem specimens, classified the posterior portion of the urethra into three types according to the relationship between the openings of the ejaculatory ducts and the prostatic utricle. Their type 2, in which the ducts open on the floor, the walls or the lips of the utricle, constituted 16.7 per cent of the specimens and is the type in which the majority

100. Zentmayer, W.: The Prostate as a Remote Focus in Ocular Infections, J. A. M. A. **87**:1172-1176 (Oct. 9) 1926.

101. Thomasson, A. H.: Chronic Gonorrheal Prostatitis, a Possible Aetiological Factor in Certain Inflammations of the Eyes, Arch. Ophth. **52**:546-553, 1923.

102. Fanz, J. I., and McCrea, L.: Anatomico-Histologic Variations in the Posterior Urethra Influencing Its Pathology and Therapy, Urol. & Cutan. Rev. **35**: 409-415, 1931.

of the complications occur. It is conceivable that some such anatomic factor contributes to a tendency to gonorrheal metastasis. In the light of the established histopathologic aspects of acute gonorrheal inflammation, the opinion of Kiene [103] regarding the frequency of metastatic gonorrheal lesions in the male is particularly logical and significant. He stated the belief that because the rich capillary plexus of the posterior portion of the urethra is damaged (phlebitis, thrombosis, etc.) early in the course of the disease, the frequent erections due to the inflammatory irritation are sufficient trauma to encourage the spread of the inflammatory process by means of these blood vessels. In women the local factors which lead to the production of metastatis are trauma to the cervix uteri as well as anatomic and physiologic changes incident to menstruation and pregnancy. The role of trauma in the production of the local as well as the metastatic complications of gonorrhea is well established. The concept that certain strains of gonococci are more prone to produce metastatic lesions has had strong adherence since the discovery of the organism by Neisser in 1879. Bumm, who contributed much to the bacteriology and histopathology of gonorrhea, as well as Jadassohn, was inclined to this opinion. Experimentally, gonococci isolated from a lesion of metastatic gonorrheal arthritis on inoculation in the human urethra produced not only specific urethritis but arthritis and pleuritis as well (Lemierre and Faure-Beaulieu [95]).

As to the second aspect of gonorrheal metastasis, the channels through which the spread takes place: It is established that gonorrheal inflammation spreads topically by surface progression and in the deeper tissues by direct continuity. However, a distant transfer, such as a transfer to the lacrimal gland, cannot originate by either of these means. The rapidity and the diversity of the occurrence of the metastasis indicates that some rapidly and more generally diffusing agent is the vehicle for the spread. Whether this agent is the blood or the lymph was earlier a controversial point. Actually it has been proved that gonorrhea may be spread by either of these means. Dreyer [104] stressed that spread by lymphatic channels in the local and in the metastatic complications of urethritis. That the dissemination of gonococci by this means may not be confined to purely local conditions is suggested by the finding of Forkner [105] of gonococci in the axillary node of a man who had previously suffered

103. Kiene, E.: Ueber die Pathogenese der gonorrhoischer Komplicationen, Arch. f. Dermat. u. Syph. **156**:663-676, 1928.

104. Dreyer: Gonorrhoe in Lymphgefässe, Arch. f. Dermat. u. Syph. **60**:259-262, 1902.

105. Forkner, C. E.: Gonococci in Material from Lymph Node, Bull. Johns Hopkins Hosp. **43**:257-259, 1928.

urethritis. Even this finding, however, may argue for a vascular spread rather than a lymphatic one. The spread of gonococci by means of the blood stream has been definitely established. Numerous investigators have recovered the gonococcus from the blood stream by culture. Classic investigations include those of Thayer and Blumer [106] on gonorrheal septicemia and of Sidler-Hugenin [23a] on metastatic ocular lesions. The work of Lemierre and Faure-Beaulieu [95] on this subject formed part of the early foundation of the study of "systemic gonorrhea." It is also established that hematogenous invasion is coincident with a definite febrile reaction.

As to the specific agent responsible for the distant inflammatory reaction: Is the gonococcus or its toxin responsible for the reaction appearing at the distant focus? Are secondary organisms responsible, or is a symbiosis of the gonococcus and some other organism the causative factor? In those cases in which no organism can be found at the site of the metastasis it has been customary to incriminate the gonococcic endotoxin as the etiologic agent. There is evidence for the belief that some of the "metastatic" appearances can be due to the toxin alone. A toxic albuminuria in association with gonorrhea is not uncommon, but it is rare to find the gonococcus in the kidney even in cases of gonorrheal sepsis at autopsy. Schieck [90] described a phlyctenular lesion in the conjunctiva in a case of gonorrhea. By experimental means he demonstrated that the lesion was an allergic manifestation to the gonorrheal endotoxin. There are authors who believe that the so-called scleroconjunctivitis of Heerfordt [83] should be regarded as a toxic manifestation, whereas in those cases of metastatic gonorrhea in which the palpebral conjunctiva is more involved this involvement should be regarded as due to the organism. Those authors who contend for the toxic basis of metastasis are confronted with the problem of explaining why it is that a virginal site should be singled out by such a generally diffusing agent as the toxin. There is more laboratory and histologic evidence that the gonococcus itself is the initial offender. Mention has already been made of the finding of the gonococcus in the pus from metastatic glandular lesions. The organism has likewise been identified histologically in tissue at the site of metastasis, e. g. in the heart valves and the endocardium, in the joints, in the skin in cases of metastatic gonorrheal cutaneous lesions, in the conjunctiva and in the capillaries of the iris. It is true that the pus found at the site of metastasis in some cases contains not only gonococci but such organisms as staphylococci, streptococci, colon bacilli, meningococci and Friedländer bacilli. There is

106. Thayer, W. S., and Blumer, G.: Endocardite ulcéreuse blénorrhagique, Arch. de méd. expér. et anat. path. **7**:701-718, 1895.

yet little valid evidence as to the exact role of these organisms in the process of metastatic gonorrhea. According to Warden [107] they may conceivably enhance the virulence of the gonococci. The general tendency is to minimize the finding of these secondary organisms. Early in the history of this subject the opinion was advanced that the metastatic lesions were due only to secondary organisms, but this concept has had little if any experimental proof and is now largely discounted.

As to those factors which favor localization of the organisms at a particular site: Rosenow,[108] speaking specifically of the streptococcus, stated that organisms carried in the blood stream are influenced to localize in certain tissues selectively by the oxygen tension of the tissues, their secretory activity and such deleterious forces as cold and trauma. There is no reason to assume that the influence of these factors is in any way peculiar to the streptococcus. It seems equally applicable to the gonococcus. Kiene [103] stated the belief that consideration must also be given to occupational trauma (as opposed to isolated violence) and past or coincident disease in the area of localization of gonococci. He emphasized the fact that anything which can affect the cellular metabolism and thereby produce a locus minoris resistentiae contributes to the selective localization of circulating organisms. The role of a previous or an existing exogenous conjunctivitis in the pathogenesis of metastatic gonorrheal conjunctivitis has been stressed by some authors. Rosenow [108] stated that certain organisms may acquire an affinity for localization in a particular site by virtue of a previous localization there by chance or otherwise. The propensity of certain strains of gonococci to do this is suggested by those experiments in which organisms obtained from inflamed joints produced gonorrheal arthritis when injected into other persons. It is conceivable that mechanical factors can play a part in the localization of infection if the organisms are circulating in the form of an infected clot or as a pus particle, but this could apply only when the locus was in or "proximal to" the lungs. It is probable that the pleuritis in "systemic gonorrhea" is on this basis. The large number of metastatic foci "beyond the lung" in gonorrhea, however, makes this factor less significant and suggests that the vascular invasion in gonorrhea is more often in the form of bacteremia than of pyemia or of septicopyemia.

The fifth aspect of metastatic gonorrhea which has a bearing on the present case is the occurrence of metastatic lesions long after the primary lesion has been quiet and the tendency to recur, often in the same locus,

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107. Warden, C. C.: The Staphylococcus and Gonorrhea, J. Infect. Dis. **13:** 124-135, 1913.

108. Rosenow, E. C.: Elective Localization of Streptococci, J. A. M. A. **65:** 1687-1691 (Nov. 13) 1915.

not infrequently after a long period of quiescence. The most logical explanation of these phenomena is the rarely demonstrated fact that gonococci possess the ability to remain localized in tissue for an indefinite period in an inactive but still viable state, capable of being stimulated to their former virulence. Dreyer [104] expressed the opinion that the organisms can remain viable in the lymph vessels for long periods. Rosenow [108] and Forkner [105] isolated viable gonococci from lymph nodes which were quiescent and not necrotic in patients whose original lesion had been "cured" years previously. In Forkner's case the primary lesion had occurred eight years before. In a case of iridocyclitis provoked by a cataract extraction, gonococci were positively identified by laboratory means according to Kravitz.[109] The primary lesion had occurred sixty years previously. This astonishing property of the gonococcus suggests that "cure" of the primary lesion is most often only an apparent one. Indeed, latent urethritis has flared up after fifty years, the flare-up following a prostatectomy (Fraser and Dye [110]). In view of these findings, it is even more justifiable to ascribe the dacryoadenitis in the case reported by de Lapersonne [38] to gonorrhea even though the urethritis had been "quiet" for about twenty years. Latent gonorrheal foci may be lighted up by trauma (Kravitz [109]) or by the specific antigen, as from a recurrence of the primary lesion or from the injection of gonorrheal vaccine (Woods [111]).

A survey of the literature suggests that gonorrheal metastasis and hematogenous invasion by the gonococcus is in general considered an unusual complication. It is this fact more than any other which has inspired much study for elucidation of the first three aspects of metastatic gonorrhea previously considered: (1) local factors affecting metastasis, (2) the metastatic vehicle (3) and the metastasizing agent. Even in the most modern textbooks one reads that "systemic gonorrhea" is only an occasional occurrence. There is, however, a concept of this subject which is much more enlightening and in keeping with the preponderance of experimental evidence and which allows for a more complete synthesis of these three seemingly isolated aspects of the subject. This is the concept which disallows the purely local nature of any type of gonorrhea, having among its advocates Wertheim and Kiene.[103] According to this concept all gonorrhea early in its course invades the blood stream. In support of this opinion is the fact that gonococci have been recovered from the blood stream by culture in

109. Kravitz, D.: Postoperative Endogenous Infections, Am. J. Ophth. **19**: 328-330, 1936.

110. Fraser, C. K., and Dye, W. J. P.: Exacerbation of Latent Gonorrheal Urethritis, J. A. M. A. **105**:269 (July 27) 1935.

111. Woods, in discussion on Carroll.[84]

cases of uncomplicated gonorrhea, in which no metastatic manifestations occurred. According to Kiene the invasion is coincident with a febrile reaction and a feeling of malaise. The fact that the manifestation of lodgment in distant foci is the exception rather than the rule suggests that the invasion is in the form of a bacteremia in which bacteria in small numbers pass rapidly through the blood stream and are phagocytosed in passage. Whatever their subsequent fate, they are in the usual case rendered inert. This accounts for the fact that successful blood cultures must be made at an opportune time as well as under optimum conditions. There is convincing evidence that even the so-called local complications of acute gonorrhea are spread hematogenously. If this concept is accepted, i. e. hematogenous invasion in almost all cases of acute gonorrhea, then the first three aspects of gonorrheal metastasis which I have discussed diminish in significance and the factors which influence the localization of the organisms in tissue and influence reactivation assume prime importance in the pathogenesis of metastatic gonorrhea.

Examination of the present case in the light of these phases or aspects of the process of gonorrheal metastasis shows, first of all, that aside from the fact that the dacryoadenitis occurred in a male there is little evidence as to the local factors which contributed to the spread of the gonorrhea. As to the second aspect, the metastatic vehicle, the organism could not be recovered from the blood stream by culture. It is felt that the failure was due to technical reasons and does not in any way contradict the metastatic nature of the disease. The extreme lability of the gonococcus in the presence of a high febrile reaction, as pointed out by Finger and associates,[69] is to be considered. The histologic picture was definitely indicative of an inflammatory process of hematogenous origin. In view of the recovery of gonococci in other metastatic processes, in the light of Sselkow's [68] demonstration of the role of the histiocyte in gonorrhea and in view of the demonstrated ability of the organism to remain localized in tissue for long periods of time, it is suggested that some of the "mast cells" observed in the microscopic sections were bacteria-containing histiocytes and that these organisms were responsible for the inflammation of the lacrimal gland.[112]

In order to evaluate properly those factors in the lacrimal gland which contribute to the localization of organisms there, one must first

112. The microscopic sections were submitted for opinion to well known pathologists, to an authority on hematology and to a histologist, all of the University of Chicago. All but one entertained the strong probability that some of the cellular inclusions were bacteria (fig. 10), with due reservations because of the inconclusiveness of histologic preparations for the demonstration and identification of bacteria in tissue.

consider the anatomy and physiology of that organ. The lacrimal gland is described as consisting of an inferior, or palpebral, lobe and a superior, or orbital, lobe. This division into lobes is brought about by the deep indentation made by the lateral horn of the aponeurosis of the levator as it extends to the lateral palpebral ligament. This indentation may be and often is so deep as to have the effect of almost isolating the so-called palpebral lobe, so that in a large number of cases it projects out into the upper-outer part of the fornix of the conjunctival sac. The blood supply to this portion of the gland comes largely through the orbital portion of the gland. There is anatomic and experimental evidence that the lacrimal gland is retracted into the orbit by contraction of the levator and returns to its relaxed position because of the resiliency of the orbital tissues against which it is pressed during retraction. Because of the fascial attachments of the gland these excursions must be small but conceivably are sufficient to affect the blood supply and physiology of the palpebral, or partially constricted, portion of the gland. The resultant of this effect would be to create a localized division of the vascular bed which would be capable of delicate and rapid fluctuation in its vascular physiology, a situation which is somewhat comparable to that of the "cut-off" muscle and the erectile tissue. The possible implications of such a delicate vascular mechanism are many. It could, for instance, well serve the rapid fluctuation in function which the lacrimal gland is called to perform in going from the resting stage to that required in reflex lacrimation and weeping, for such a vascular arrangement could cause a localized high rise in the capillary pressure, which is in keeping with rapid secretory activity. In this sense it is somewhat analogous to the relationship of the intraocular capillary pressure to the production of aqueous. Treacher Collins,[113] in his Mackenzie Lecture on the physiology of weeping, placed great importance on the movements of the lids and their influence on the blood supply and the function of the lacrimal gland.

It has been found that the lacrimal gland is subject to great variation in its anatomy. Whitnall [114] mentioned the occurrence of a muscular slip from the levator which was attached to the orbital gland and served to retract the gland appreciably when the levator contracted. Other reduplications of various fascial strips about the gland have been described as ligaments of the gland. When such anatomic peculiarities are present it is conceivable that the seclusion, or "embarrassment," of the vascular function is even greater than normal and that as a result the range of safety for fluctuation in the blood supply, rate and pressure,

113. Collins, E. T.: Physiology of Weeping, Brit. J. Ophth. **16**:1-20, 1932.

114. Whitnall, S. E.: Anatomy of the Human Orbit, London, Oxford University Press, 1932, p. 208.

in the oxygen tension, etc., is a narrow one. These anatomic variations may well permit all the changes which the lacrimal gland is required to undergo under physiologic conditions but still render its blood supply incapable of adjusting to the great fluctuations required in a toxic state, so that pathologic change results when this mechanism is taxed too greatly. Such a lowering of the nutrition of this part of the gland under inflammatory conditions makes it a suitable nidus for the lodgment of any organisms circulating in the blood stream at the time. That anatomic variations of a type or degree sufficient to create this chain of circumstances are apparently rare accounts for the fact that metastatic dacryoadenitis is uncommon. Such a concept also allows for the rare unilateral occurrence of the condition.

It is felt that such were the conditions which led to the first occurrence of the patient's dacryoadenitis four years previously. As for the second attack, organisms remaining in the gland which were deposited at the time of the initial infection would be liable to activation by the toxins from the immediately preceding urethritis. The time interval in this instance was four years, which is not remarkable when one considers the case in which Forkner [105] isolated gonococci after eight years or the case in which Kravitz [109] did so after sixty years. The evidence for the influence of this factor, the presence of organisms in tissue, on the third and fourth attacks is even stronger. The influence of temperature on the second attack, which occurred during a period of unusually cold weather, is also to be considered, for cold is given as a cause of dacryoadenitis by Elschnig,[26a] and Casali [54] stated the belief that cold was a contributory factor in the case of gonorrheal dacryoadenitis reported by him. In the fourth attack the circumstances differed from those in any of the previous attacks. The patient denied any sexual contact or use of alcohol prior to the increase in the ocular symptoms. But he did receive an injection of gonococcic vaccine, which caused a violent reaction at the site of the injection. It is evident that this injection was the cause of the exacerbation of the lacrimal disease. Urethral symptoms were increased coincidently and not previously, as in the other attacks. In my opinion, then, the pathogenesis in this instance was the activation of the organisms in the tissue by the gonorrheal antigen. Such a focal reaction from the injection of gonorrheal vaccine in a case of conjunctivitis was reported by Woods.[111]

Pincus [88] in a case of metastatic gonorrhea described a wrinkling of the corneal epithelium such as was seen during the first few days of disease in the present case. Nosologically the corneal lesions observed during the second recurrence were lesions of superficial punctate keratitis, but there is little to indicate their significance for metastatic gonor-

rheal disease. They were neither the superficial punctate erosions nor any of the raised punctate lesions such as are sometimes found in the cornea in gonorrheal conjunctivitis. Whether they were due to the disease process or to the local therapy is a question.

IV. SUMMARY AND CONCLUSIONS

The history, clinical picture and etiologic aspect of acute dacryoadenitis in general and of gonorrheal dacryoadenitis in particular are described and discussed. Gonorrheal dacryoadenitis is infrequent in incidence, only 39 cases having hitherto been reported. These are found in 26 case reports.

A case is presented of acute bilateral dacryoadenitis of metastatic gonorrheal origin in which there were two recurrences during observation and in which there was a clearcut history of a similar disorder four years before.

Recurrence in dacryoadenitis from any cause is apparently the most unusual aspect of the disease. This is especially true of gonorrheal dacryoadenitis, since only 1 such instance has been reported previously, that of Seeligsohn.[8] The present case is the first reported in which there was more than one recurrence; there were three in all (four attacks, all bilateral).

The present account of the histopathologic process in a portion of the gland removed for biopsy is the first detailed description of the histopathologic picture of gonorrheal dacryoadenitis in the available literature. The observations make evident the fact that the gonorrheal inflammatory process in the lacrimal gland is the same as that of gonorrhea in any other tissue and support the view that metastatic gonorrhea is basically the same as primary gonorrhea, being modified only by the anatomy and physiology peculiar to the involved organ.

The complication in this case of gonorrheal dacryoadenitis by the formation of dacryops is the first such complication reported. It was demonstrated by the microscopic study that the dacryops formation was located in an extralobular excretory duct and consisted of a dilatation of this duct. Such dilatation is the resultant of more than one factor, the primary ones being destruction of the mechanism which maintains the contractibility and the support of the ducts by pericanalicular infiltration of inflammatory cells and by toxins. The blockage, which may be incomplete, of these ducts by mucus and cellular exudate is under these circumstances sufficient to cause the ducts to become dilated by the relatively low secretory pressure of the lacrimal gland.

The clinical and histologic evidence seems to indicate that the inflammation of the gland is due to the actual presence in the gland tissue of

the specific organisms. These organisms reach the gland through the blood stream and once deposited set up an interstitial inflammation. The chief factor which contributes to the selective localization of these organisms in the gland is anatomic, perhaps a congenital anomaly which serves to restrict further the already normally isolated blood vascular system of the palpebral portion of the gland.

Recurrences and focal reactions, which were a striking feature of this case, are due to the little emphasized but proved fact that gonococci possess the peculiar property of remaining viable in tissues, even though inactive, for remarkably long periods of time (sixty years [109]) and capable of being reactivated by noxious stimuli, such as trauma, cold or the presence in the blood stream of the specific toxin.

From the histologic evidence of early occlusive vascular lesions in gonorrheal dacryoadenitis, it would seem that vasodilatory therapy is a logical adjunct to "specific" therapy (use of sulfanilamide and its derivatives) in the acute phase of the disease.

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News and Notes

EDITED BY DR. W. L. BENEDICT

GENERAL NEWS

Pan-American Congress of Ophthalmology.—Through the W. K. Kellogg Foundation, of Battle Creek, Mich., the Pan-American Congress of Ophthalmology announces the establishment of an international graduate student exchange. With the cooperation of the Department of State of the United States, twenty-five graduate physicians from Latin America will be brought to the United States for graduate training in ophthalmology. They will be assigned to seventeen of the leading ophthalmic institutions to serve as accessory residents for a minimum of one year and longer if they show unusual ability. The traveling expenses to and from the country of origin will be paid, and each man will receive $1,000 a year from the foundation.

The Latin American physician will be chosen in each country by a committee composed of the dean of the medical school, the professor of ophthalmology and an already existing committee of the American legation, under the supervision of the Department of State. The number of scholarships allotted to each country will be based on the population and on the number of ophthalmologists in the country. Applications must be filled through the local professor of ophthalmology and forwarded to the secretaries of the Pan-American Congress of Ophthalmology. Those south of Panama are to be sent to Dr. M. Alvaro, São Paulo, Brazil, and those north of Panama, to Dr. Conrad Berens, New York city. Each application must contain full information regarding the candidate and must bear an exhaustive endorsement by the professor of ophthalmology. After preliminary consideration by the secretarial board of the Pan-American Congress of Ophthalmology, the applications will be returned to the previously mentioned local committees for selection of candidates. All the information about the selectees will be sent by the local committees through the Pan-American Congress secretaries to the foundation for final acceptance.

Eligible for such scholarships are Latin American physicians who are natives of the country in which their applications originate. The applicant must be less than 30 years of age, must be a graduate of a recognized medical school, must have served at least one year as a rotating intern or as assistant in a medical clinic or have had at least three years of general practice of medicine, must be particularly interested in ophthalmology, must have a good working knowledge of the English language, must agree to return to his home country to practice and teach ophthalmology on completion of his scholarship and must agree to submit to the usual discipline of the institution to which he is assigned. Furthermore, he must agree to attend at least one and preferably two meetings of any of the national societies of ophthalmology of the United States.

The ophthalmic institutions accepting the Latin American graduates have agreed to extend to them the same training that is given to their

own residents, with the single exception that in some of the clinics physicians not licensed in the United States are not permitted to perform operations on patients. However, they may assist.

Applicants will be assigned to the ophthalmic institutions by the Pan-American Congress of Ophthalmology committee as they are accepted. No consideration will be given to requests for assignment to specific clinics. The work will start as soon as transportation can be arranged. The following ophthalmic institutions are participating: the Massachusetts Eye and Ear Infirmary, Boston; the Ophthalmic Institute of Columbia University College of Physicians and Surgeons, the Bellevue Hospital, and the New York Eye and Ear Infirmary, New York; Wills Hospital, Philadelphia; Wilmer Ophthalmological Institute, Johns Hopkins Hospital, Baltimore; University of Michigan Medical School, Ann Arbor; University of Cincinnati College of Medicine, Cincinnati; Northwestern University Medical School, Illinois Eye and Ear Infirmary and Cook County Hospital, Chicago; the Mayo Clinic Rochester, Minn.; State University of Iowa College of Medicine, Iowa City; Washington University School of Medicine, St. Louis; Tulane University of Louisiana School of Medicine, New Orleans, and Stanford University School of Medicine and University of California Medical School, San Francisco.

Pray and Burnham Prizes.—These prizes are awarded annually to the authors of the two best essays submitted by members of the New Hampshire Medical Society during the preceding year. They are announced at the annual meeting of the New Hampshire Medical Society by its trustees. The first prize, of $100, was this year given to Dr. Arthur Linksz, of the Dartmouth Medical School Eye Institute, in Hanover, for an essay entitled "Dermatotherapy as Applied to Diseases of the Eyelids," as announced May 13.

Howe Medal.—At the meeting of the American Ophthalmological Society at Hot Springs, Va., June 1 the Howe Medal was awarded to Dr. E. V. L. Brown, of Chicago.

SOCIETY NEWS

Reading (Pa.) Eye, Ear, Nose and Throat Society.—The eleventh meeting was a joint meeting with the Reading Dental Society. It was held May 20 at the Hotel Abraham Lincoln. There were eighty-five present: Fifty-four dentists, twenty-two ophthalmologists and otolaryngologists and nine guests. The meeting was addressed by Dr. Robert Henry Ivy, professor of maxillofacial surgery at the University of Pennsylvania School of Medicine. The subject of his talk was "Plastic and Maxillofacial Surgery." The subject of the next meeting of the organization will be "Eye, Ear, Nose and Throat Aspects of Chemical Warfare."

Ophthalmological Society of the United Kingdom.—The annual meeting was held May 29 and 30, and the following subjects were discussed: "The Value of Sulfonamides in Ophthalmology" and "The Role of Vitamins in Ophthalmology."

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

ERRONEOUS CONCEPTION OF MÜLLER'S FIBERS. E. P. FORTIN, Arch. de oftal. de Buenos Aires **15:** 325 (July) 1940.

Basing his views on his entoptic studies of the retina, and on his consideration of the usual descriptions and illustrations of this structure as derived from cadaveric retinas, Fortin calls special attention to the errors found in connection with Müller's fibers as the sustaining neurologic elements, most pronouncedly in Ramon y Cajal's and Kolmer and Lauber's publications.

In these publications, this so-called sustaining network is depicted as a coarse network of giant epithelial cells situated, at variance with all histologic tenets, in the midst of a vascular layer, the thickness of the fibers in relation to those of the remaining retina being in the proportion 1 to 6. If this were the case vision would have to be effected through a grill so thick as to interfere greatly with its accuracy.

In entoptic observation, by Hardinger's method among others, the structure of the retina appears beautifully and regularly arranged, with a fiber thickness of 1 micron in contrast to the 50 microns of Cajal's preparations, the layers corresponding to Henle's division into an external neuroepithelial and inner cerebral layer.

The same contrast is seen in the limiting membrane, external and internal, the former being the best defined membrane of the body, with a uniform thickness of 1 micron, the orifices through which pass the rods and cones being clearly observable by entoptic observation.

So-called Müller's fibers are artificially produced canals in the gelatinous structure of the retina.

This paper is illustrated with numerous photomicrographs from the authors and from Ramon y Cajal's publications. C. E. FINLAY.

Conjunctiva

PRIMARY TUBERCULOUS INFECTION OF THE CONJUNCTIVA. I. MALDONADO ALLENDE and M. JOISEN, Arch. de oftal. de Buenos Aires **15:** 625 (Dec.) 1940.

A case is reported of primary tuberculous infection of the conjunctiva accompanied with inflammation of the corresponding submaxillary lymph gland. Tests for syphilis gave negative results; the blood count and the urine were normal; the reaction to the Mantoux test was at first negative and later 4 plus. Examination disclosed no germs in the conjunctival secretion, and cultures were negative. Lymphoid and epithelial cells were noted. Improvement was obtained with dietetic and

hygienic treatment; administration of calcium, vitamins A, C and D and methyl antigen, and heliotherapy. The differential diagnosis in relation to sporotrichosis, Parinaud's conjunctivitis, conjunctival chancre and tularemia is discussed as are the prognosis and treatment.

C. E. FINLAY.

PRIMARY TUBERCULOSIS AND LUPUS OF THE CONJUNCTIVA. S. TALKOVSKIY, Vestnik oftal. **16:** 212, 1940.

Talkovskiy discusses the etiology and pathogenesis of lupus and the differential diagnosis of lupus and primary tuberculosis of the conjunctiva. The rarity of tuberculosis of the skin can be explained by the low temperature of the skin and its light vascularization. The eye is involved in about 88 per cent of cases of lupus of the face; the skin of the lids is more frequently involved than the conjunctiva.

Two cases are reported. In one the lid was ptosed and the conjunctiva of both lids was thickened with a number of papillae. There were scars on the skin near the nostrils—healed lupus. Histologic examination showed granulation tissue of specific structure. Ziehl-Neelsen staining showed acid-fast bacilli. In the second case there was lupus of the face and of the conjunctiva of the left eye. Roentgen ray treatment did not improve the process.

The tuberculosis of the conjunctiva in both cases was secondary, because there was no involvement of the preauricular gland and no necrosis of the tissue. Talkovskiy stresses the point that though the etiologic agent in both lupus and primary tuberculosis is the acid-fast bacillus, lupus and primary tuberculosis of the conjunctiva are independent nosologic units.

O. SITCHEVSKA.

Comparative Ophthalmology

REPRESENTATION OF THE SYMPATHETIC AND PARASYMPATHETIC NERVOUS SYSTEM IN THE FOREBRAIN OF THE CAT. H. B. CARLSON, E. GELLHORN and C. W. DARROW, Arch. Neurol. & Psychiat. **45:** 105 (Jan.) 1941.

"The excitation of various parts of the forebrain of the cat with faradic currents and the recording of the reaction of the pupil (normal and sympathectomized), the nictitating membrane, the blood pressure and the galvanic reflex have shown that parasympathetic inhibition, as indicated by pupillary dilatation of equal degree in the normal and in the sympathectomized eye, may be obtained from the corona radiata, the basal ganglia and the hypothalamus and pretectal area. In the hypothalamus this response may be associated with signs of sympathetic excitation.

"In general it has been found that the intensity of the autonomic response increases as the electrode descends from the corona radiata through the thalamus into the hypothalamus.

"From the pretectal region constriction of the pupil is elicited, followed by dilatation (inhibition of parasympathetic control).

"Parasympathetic and sympathetic responses may appear in various combinations, indicating that the excitation or inhibition of one branch of the autonomic nervous system does not determine the nature of the excitatory process in the other, functionally opposed division."

R. IRVINE.

Cornea and Sclera

MARGINAL DYSTROPHY OF THE CORNEA. J. A. PEYRET, Arch. de oftal. de Buenos Aires **15:** 459 (Sept.) 1940.

Two cases of marginal dystrophy of the cornea are reported. One patient was an Argentine and the other a Spanish woman. In one case, of unilateral involvement, personal and familial history was noncontributory, the evolution was torpid and there were some corneal thinning and ectasia which were finally brought to a standstill. The cases are illustrated by photomicrographs, and the slit lamp observations are detailed. A general consideration of the symptoms, pathologic changes, etiologic factors, diagnosis, evolution and treatment follow the case reports, with a detailed review of the literature accompanied by a comprehensive bibliography.

C. E. FINLAY.

RING-SHAPED CORNEAL PRECIPITATES: DEVELOPMENT OF MIGRATING TUBERCLES AT THE INTERIOR SURFACE OF THE CORNEA; REPORT OF A CASE. E. BRUENS, Klin. Monatsbl. f. Augenh. **104:** 311 (March) 1940.

A man aged 36 had noted paresis of the facial nerve on the right side and later on the left side about six months prior to observation. His eyes burned and were closed by exudate, and the vision was decreased at times. During the preceding month obscurations of the vision had appeared. Iridocyclitis and a number of ring-shaped precipitates on Descemet's membrane were observed. A roentgenogram of the lungs showed considerable enlargement of the hilus but no foci of recent infection in the lungs. A small dental granuloma was extracted. The sinuses were considered normal. A blood culture was negative for tubercle bacilli. The reaction to tuberculin (Mantoux) and the Wassermann reaction were negative. The percutaneous reaction to ektebin (Moro's tuberculin in an ointment base containing a keratolytic substance) was slightly positive. No therapeutic results were obtained in the eyes. A number of photographs illustrate the article. Bruens diagnosed tuberculous iridocyclitis. The negative result of two intracutaneous tuberculin tests was in the author's opinion a sign of anergy of the organism. Five morphologic stages of the precipitates are distinguished and described. The development of the rings out of originally homogeneous precipitates could be followed. The rings formed as residues of a tuberculous nodule harbored in a fluid medium. A special property of the nodule was required to form the rings. This property the author believes to comprise the rapid growth of the anergic organism of the patient in the tubercles with emptying of the degenerated central

portion into the anterior chamber. Subsequent degenerative substances were flushed off by the aqueous humor, but firmer cell formations at the periphery remained, forming the rings. K. L. STOLL.

General Diseases

OCULAR MANIFESTATIONS IN PORADENITIS. J. MALBRÁN and E. J. LIMA, Arch. de oftal. de Buenos Aires **15:** 524 (Oct.) 1940.

After a brief review of existing knowledge of poradenitis, known under different names (inguinal lymphogranuloma, benign lymphogranuloma, venereal lymphogranuloma, poradenitis, poradenolymphitis, Nicholas Favre's disease), the authors report their investigations in 24 cases in connection with the ocular symptoms. Apart from local conjunctival lesions at times simulating Parinaud's conjunctivitis and indicating an extragenital infection, they found papillary edema with blurring of the disk margins and dilatation and tortuosity of the neighboring vessels in 82.6 per cent of the cases (with no noticeable loss of sight), pointing to a septicemic process. C. E. FINLAY.

ARACHNODACTYLY AND BILATERAL ECTOPIA OF THE LENS. M. KRASNOV, Vestnk oftal. **18:** 198, 1941.

A woman aged 50 had acute glaucoma of the left eye with the lens luxated into the anterior chamber. In the right eye the lens was subluxated down and in. Her appearance was typical of arachnodactyly. She was tall and thin, with spider-web fingers and a narrow pectinate chest. The luxated lens was extracted from the anterior chamber, after which the eye became quiet and the tension normal, but the vision was only perception of hand movements. O. SITCHEVSKA.

Glaucoma

TYPES OF GLYCEMIC CURVES AND THEIR CLINICOPATHOGENIC VALUE IN GLAUCOMA. M. POPOV, Vestnik oftal. **18:** 161, 1941.

This study was made (1) to demonstrate the glycemic curves in glaucoma as reflected by the general neuroendocrine "background" predisposing to glaucoma and (2) to evaluate the clinical course of the glaucomatous process in its pathogenetic relation to the types of glycemic curves.

The methods were as follows: 1. An aqueous solution of dextrose was given orally, 1 cc. per kilogram of body weight (277 patients). 2. With oral administration of dextrose, 0.3 cc. of epinephrine hydrochloride (1:1,000) was injected (70 patients). 3. The method of Staub and Trayhott, specific for the pancreas, was utilized (90 patients). The blood sugar was determined every fifteen minutes for two and one-half hours after fourteen hours of fasting. There were 98 patients suffering from primary glaucoma. The control group consisted of 66 patients with senile cataract and 53 patients with trachoma, corneal ulcers and iridocyclitis. The curves could be divided into four types

(except the diabetic), the profiles which are given in tables. The relation of the clinical course of glaucoma with the glycemic types of curves was as follows: An acute form of glaucoma—loss of vision, headaches, the seeing of rainbow colors, etc.—was observed with the first type. With the other types were observed milder forms of glaucoma or simple glaucoma. Thus it could be assumed that with the first type the basis of the pathogenetic mechanism of glaucoma lies in the general sensitivity of the sympathetic-adrenal system and also of the cervical portion of the sympathetic ganglion, which is connected with the ciliary ganglion; this theory could explain also the acute form of glaucoma due to circulatory disturbances.

Popov appeals to ophthalmologists to make a further study of the complicated relation of the eye and the adrenals, pancreas and liver.

O. SITCHEVSKA.

Injuries

GLASS IN THE ANTERIOR CHAMBER. T. C. SOMMERS and H. HOBBS, Brit. J. Ophth. **26:** 54 (Feb.) 1942.

The authors' reasons for reporting their case were to emphasize the severity of the irritation caused by glass in the anterior chamber, to confirm the possibility of its removal once it has been located and to record that full recovery of vision is possible after removal. The spicule of glass entered the eye as a result of injuries received during an air raid. The presence of the glass was noted at the time of the injury, but its removal was then impossible. The eye rapidly recovered, with excellent vision. About five months later, severe inflammation set in, and vision was reduced to light perception. After local treatment had cleared the cornea, the foreign body could be seen lying in the lower angle of the chamber. It was decided to do an iridectomy, but on insertion of the blades of a Barraquer forceps the glass was immediately felt, and it was removed without further manipulation. The resulting visual acuity was 6/5.

W. ZENTMAYER.

MUCOUS MEMBRANE TRANSPLANTS IN TREATMENT OF CORNEOCONJUNCTIVAL BURNS. G. VON GROLMAN, Arch. de oftal. de Buenos Aires **15:** 430 (Sept.) 1940.

A case is reported in which a deep, severe burn with sodium hydrate involving the bulbar conjunctiva, the inferior half of the episclera and the cornea, with clouding and grayish infiltration of the cornea, was treated on the day of the accident by free excision of the cauterized tissue and application of a transplant from the mucous membrane of the lip. After ten months there were complete cicatrization of the affected conjunctiva and episclera without any retraction and complete clearing of the corneal opacity. The site of the transplant remained, however, decidedly red and much thicker than the surrounding conjunctiva.

Von Grolman reviews the literature on the treatment of burns of the conjunctiva by immediate excision and transplantation of mucous membrane, the method first advocated by Denig in 1912, with the object of prompt elimination of the caustic agent, which continues acting in

the deep tissues as long as it is present, and prevention of retracted cicatrices, symblepharons, etc. He next discusses the nature of the transplants used: living human conjunctiva, epidermis, genital mucous membrane, labial mucous membrane, cadaveric conjunctiva, fetal conjunctiva and yolk of egg, which can be utilized under certain circumstances.

C. E. FINLAY.

Lens

SUPERFICIAL EXFOLIATION OF THE LENS CAPSULE (VOGT'S CAPSULAR GLAUCOMA). J. MALBRÁN and B. TOSI, Arch. de oftal. de Buenos Aires **15:** 357 (July); 407 (Aug.) 1940.

In this paper new explanatory characteristics are described and former views brought forward in connection with Vogt's capsular glaucoma.

The priority of the description, usually accorded to Vogt, belongs to Lindberg, a fact which does not detract from the remarkable contribution of the former.

The triad of symptoms which Vogt held to indicate a diagnosis (the disk on the center of the lens surface, peripheral exfoliation and deposits on the pupillary margin) is beyond discussion. Malbrán and Tosi, however, explain in more detail the changes in the central, the intermediate and the peripheral zone of the capsule as seen with high magnification and with mydriasis, and they call attention to a similarity in the deposits on the iris margin to those found in the zonula and the hyaloid membrane, the latter structures having been observed chiefly through operative colobomas of the iris.

They review the prevailing views on the pathogenesis, being inclined to Kirby's opinion that the exfoliation and the glaucoma depend on a degenerative process in the endothelium at the iris angle.

They consider the prognosis of glaucoma less favorable if there is lenticular exfoliation than if there is not.

As to treatment, they agree with Kirby in considering extraction of the lens as hazardous, preferring cyclodialysis.

Eleven case reports, with illustrations, follow the main body of the article.

The paper is accompanied by an extensive bibliography.

C. E. FINLAY.

CONGENITAL ANNULAR CATARACT. J. MALBRÁN and B. TOSI, Arch. de oftal. de Buenos Aires **15:** 543 (Nov.) 1940.

Few cases of congenital annular cataract have been reported, the first reports having been mere presentations of anatomic observations and the others, with the exception of that of Hess, clinical reports. The literature is reviewed in detail and a case reported with clinical, slit lamp and microscopic (postoperative) details. Malbrán inclines in part to von Szily's views in considering the lesion a central cataract due to an idiokinetic disturbance and in part to Collins', which attribute the lesion to interruption in the second stage of the development of the

lens, with resulting nonformation of a central nucleus leading to a central membranous formation produced by apposition of the anterior and the posterior lens capsule and separating two peripheral lenticular masses which develop in a more or less disorderly fashion.

C. E. FINLAY.

Methods of Examination

INVESTIGATION OF LIGHT PERCEPTION AND PROJECTION WITH A FILIFORM RAY. F. PAEZ ALLENDE, Arch. de oftal. de Buenos Aires **15:** 641 (Dec.) 1940.

A method of investigating cataractous eyes with regard to light perception and projection by means of a filiform ray is described.

C. E. FINLAY.

NEW METHOD OF TESTING THE TYPES OF ABNORMAL COLOR SENSE BY MEANS OF COLORED DOTS: REPORT OF CASES. W. TRENDELENBURG, Klin. Monatsbl. f. Augenh. **104:** 473 (May) 1940.

The known methods of testing the color sense have some deficiencies. The Stilling-Hertel tables, for example, give the result not only for the color sense but for the form sense and the combination ability. The author has devised a method of testing solely the color sense. Disks 1 cm. in diameter are cut out of colored blotting paper. Eighteen colors are represented: red, red-orange, orange, yellow, yellowish green, green, green-blue, greenish-blue, blue-green, blue, blue-violet, violet, violet-purple, red-purple, red-blue, brown, grayish brown and gray. The proper mixture of colors and their dilution to produce the shades are described.

The person being tested points out the colored disks corresponding to disks stained with dilutions of the stock shades. Various forms of color perception may be demonstrated with this method, from the normal to weak: protanopia, deuteranopia, total color blindness, night blindness and acquired deficiencies.

A number of cases are reported. The colors are named and distinguished by numerals. Experiences with tritanopic forms have not been collected, and the exact borderline between perfect and faulty color sense will have to be designated.

K. W. STOLL.

Neurology

CHANGES IN THE BRAIN IN ALCOHOLISM. L. D. STEVENSON, L. E. MCGOWAN and A. M. ALLEN, Arch. Neurol. & Psychiat. **45:** 56 (Jan.) 1941.

The clinical histories, the general autopsy observations and the changes in the brain in 44 cases of chronic alcoholism were examined. In 22 cases the clinical diagnosis was alcoholic encephalopathy. In 18 of these there was some change in the pupillary reflex, inequality

of the pupils or paralysis of ocular muscles. In 8 there was nystagmus. The conclusions of some previous authors with regard to frequent and severe involvement of the optic nerves were not confirmed.

R. IRVINE.

RHYTHMIC MYOCLONUS: A CLINICAL REPORT OF SIX CASES. J. P. DOBSON and H. A. RILEY, Arch. Neurol. & Psychiat. **45:** 145 (Jan.) 1941.

"Rhythmic myoclonus may be described as a regularly recurrent muscular contraction, without opposing movements of antagonistic muscles. It is to be distinguished from nystagmus, in which slow motion of one muscle group is followed by a quick rectifying motion of an opposing muscle group, and from tremor, in which antagonistic groups of muscles contract irregularly and in opposite directions. It also differs from both of these and from all other myoclonic movements in its nearly perfect rhythmicity and its persistence during sleep. It can temporarily be suspended by voluntary contraction of the affected muscles.

"It is observed most frequently in muscles of the oropharynx but is also found in muscles of the face and eye, the larynx, the diaphragm and the intercostal muscles and more rarely in the muscles of the upper extremities. . . ."

The clinical findings in 6 cases of rhythmic myoclonus are presented.

"All the patients were relatively advanced in years; all but 1 showed definite arterial hypertension and evidences of extensive arteriosclerosis, both general and cerebral. All patients presented evidence of involvement of the brain stem, principally disturbances in the control of ocular movements, loss of conjugate gaze, weakness of one or the other external rectus muscle, facial palsy or bilateral involvement of the larynx. Some disturbance in superficial or deep sensibilities was determined in all the patients who were capable of sufficient cooperation to supply adequate information. Either one or both pyramidal tracts had been involved, as indicated by the history or determined by clinical examination. Only 1 patient presented a positive Wassermann reaction of the spinal fluid. The distribution of the rhythmic myoclonic movements differed in each case, except in 2 in which the characteristic movement was shown only in the posterior pharyngeal wall. In all cases the involuntary movement was inhibited by voluntary innervation."

R. IRVINE.

EXPERIMENTAL STUDIES ON HEADACHE. G. A. SCHUMACHER and H. G. WOLFF, Arch. Neurol. & Psychiat. **45:** 199 (Feb.) 1941.

In about half of the patients having migraine headaches, preheadache symptoms occur. They are commonly visual, such as scotomas, hemianopia, scintillations and other crude hallucinations of light and color. Of these scotomas are the most readily investigated.

The authors were afforded an opportunity to study scotomas because of their occurring in a physician who could make accurate and rapid observations of his own visual defects.

The effects on the preheadache scotomas of inhaling a small amount of amyl nitrite are presented in a series of visual field studies, in one

experiment when the amount of amyl nitrite inspired was insufficient to cause a drop in blood pressure or "light headedness" and in a second experiment when a larger amount was inspired, causing an abrupt fall in blood pressure and amblyopia and faintness.

From the experiments performed the investigators consider it apparent that "cerebral vasodilation associated with a sustained normal level of blood pressure caused symptoms to disappear, whereas a procedure that decreased cerebral blood flow caused the symptoms to become worse. From this it may be deduced that cerebral vasoconstriction was responsible for the visual defect in this patient with migraine. It is also likely that the cause of the visual defect was not in the retina or orbit but within the cranial cavity.

"There are several reasons for making the latter inference. First, during an attack there was sparing of central vision. Second, homonomous quadrantic defects could readily result from a single defect in the visual cerebral cortex, whereas it would be necessary to postulate a bizarre arrangement of multiple defects in the retina. Third, the progression of the scotomas from center to periphery also suggests cortical involvement, since such a defect could result from occlusion of the branches of the posterior cerebral arteries, causing ischemia of the most peripheral portions of the artery's blood supply, namely, that area near the occipital pole and spreading ultimately to involve the areas more anterior. If the defect resulted from retinal ischemia the opposite would hold. Hence, it is inferred that visual preheadache phenomena result from dysfunction of cerebral vessels, in contrast to the headache phenomena which result from dilatation of extracerebral vessels.

It is of interest that the preheadache visual phenomena seldom overlap the headache phenomena, or at most by but a few minutes. This indicates that cerebral vasoconstriction has terminated before extracerebral vasodilatation has begun. It is conceivable, to be sure, that there is also dilatation of cerebral vessels following the vasoconstriction, but it is likely that such cerebral vasodilatation plays but a slight part in the headache. . . .

"Further relevant data come from the case descriptions of Adie. He described a series of patients with long-standing periodic headaches preceded by defects in the visual fields. Such defects usually were of relatively short duration and left the patient free from symptoms between attacks. However, in each instance, after years of short-lived attacks, the patient finally failed to recover, and a permanent hemianopic defect resulted. It may be inferred from the data of these experiments that permanent tissue damage resulted either from thrombosis in a constricted cerebral artery or from too prolonged ischemia." R. IRVINE.

ENCEPHALOGRAPHY IN THE DIAGNOSIS OF CHIASMATIC AND HYPOPHYSIAL TUMORS. M. BALABO and M. F. ORIBE, Arch. de oftal. de Buenos Aires **15:** 377 (Aug.) 1940.

After the probable existence of a hypophysial tumor is inferred from the ophthalmologic, radiologic and endocrinologic data, the indicated surgical procedure requires a precise determination of its size and location in order that the most appropriate route may be selected.

Formerly Balado employed for this purpose ventriculography with iodized oil, but since his experience with diabetes insipidus he has used encephalography after injection of air by lumbar puncture (once the nonexistence of papilledema has been established) followed by spectroscopic examination of roentgenograms, the order of his procedure being as follows: (1) determination of the nonexistence of papilledema, (2) lumbar puncture with extraction of the maximum amount of cerebrospinal fluid and injection of an equal volume of air, (3) placing of the patient in Rose's position, (4) taking of a lateral spectroscopic roentgenogram of the region of the sella, (5) taking of an anteroposterior spectroscopic roentgenogram with the patient in the dorsal position with the occiput resting on the plate and (6) spectroscopic examination of the roentgenograms.

Four case histories follow the main body of the article; these are illustrated with visual field charts, encephalograms, drawings of the operative fields and two histologic plates.

A bibliography is appended.

C. E. FINLAY.

Pharmacology

OPHTHALMIC STUDY OF ALTERATIVES OF PERIPHERAL CIRCULATION. NICOTINIC ACID. G. VON GROLMAN and E. ANGEL, Arch. de oftal. de Buenos Aires **15:** 559 (Nov.) 1940.

The ocular fundus lending itself so well to the study of peripheral vascular changes and a circulatory element playing such an important part in a large proportion of ocular diseases, the authors decided to study the effect on the eye of drugs influencing the peripheral circulation, commencing with a preliminary study of nicotinic acid.

After reference to recent studies of this drug, they record its action in 23 cases, in 20 of which the tolerance was complete, in 2 of which there was some bradycardia and in 1 of which there was absolute intolerance. The intolerance may appear as excessive parasympathetic reactions (bradycardia, sweating, tremors and gastric symptoms) or an unfavorable vascular reaction (hypertension). The authors record in tabular form the effects on the pulse rate in 6 cases, in which there was a slight tendency to its increase, generally with some hypotension; the general arterial tension in 17 cases, in which there was little effect except a slight tendency to a reduction of the systolic pressure (this was rarely increased); tonometric readings in 17 cases, of which there was a slight increase in 8, a reduction in 11 and no change in 11; ocular tonoscopic readings, which disclosed in the majority of the cases a reduction of both the systolic and the diastolic retinal tension, in rare cases an increase and in exceptional cases no change; visual field changes in 7 cases, of which there was no change in 3, an increase in the 5 degree isopter in 3 and glaucoma in 1. Charts are presented, as well as roentgenograms, which seemingly show some retinal vessel dilatation. Micrometric measurements will appear in a subsequent paper.

C. E. FINLAY.

Retina and Optic Nerve

OPTIC NERVE ATROPHY FROM THORACIC COMPRESSION (PURTSCHER'S DISEASE). J. MALBRÁN, Arch. de oftal. de Buenos Aires **15**:435 (Sept.) 1940.

Sudden compression of the thorax or abdomen has frequently given rise to severe ocular lesions, which may or may not be accompanied by lesions elsewhere in the body. The first observations were on hemorrhagic lesions, usually in the retina; examples of optic nerve atrophy are much rarer. The two sets of lesions have the same pathogenic cause and according to Malbrán should be described as part of a similar syndrome: the syndrome of the posterior pole of the eyeball produced by nonocular traumatism.

The author reviews at length the literature on the subject since the publication of Jacobi's first case in 1868, most cases having been reported as instances of Purtscher's disease. The etiologic factor in 72 cases was compression of the trunk (thorax, abdomen and spinal column), in 26 cases cranial injury, in 3 cases simultaneous bodily and cranial lesions and in 1 a bullet wound in the neck.

The ocular syndrome is always due to distant trauma not directly involving the eyeball, generally of a violent character, such as that associated with falls and railway and automobile accidents. The objective symptoms are more characteristic than the subject and fix the nature of the syndrome. Occasionally conjunctival and palpebral ecchymoses are noticed, as well as ecchymoses in the neck, face and distant regions. Pupillary alterations also occur, but it is the ophthalmoscopic picture which defines the disease. This consists of retinal hemorrhages, venous dilatation, white spots of infiltration and edema. Optic nerve atrophy is much rarer, and except in 1 of Vogt's cases, in which it was preceded by choking of the disk, it came on in primarily normal disks. When it occurs there is a possibility of hemorrhage into the nerve trunk or within its sheaths.

Malbrán next takes up the mechanism of production of the disease, discussing the different theories: that of Purtscher and Vogt, which is generally accepted and which ascribes the condition to the sudden increase of intracranial pressure; that of Stahli, Best and Marchesani, which involves compression of the ocular tissues against the orbital walls, and that of Urbank and Löwenstein, which explains the lesions as a result of a fatty embolus. It is to the last theory that the author is most inclined.

A case is reported in which visual disturbances were noted after the patient, a fireman, was crushed in the overturning of a fire wagon. In the right eye optic nerve atrophy accompanied by retinal edema with hemorrhages, white spots and venous dilatation was noted. The left eye showed similar retinal lesions, with great visual reduction from central scotomas: Malbrán considers this a typical case of Purtscher's disease with final optic nerve atrophy in one eye and confirms his prior statement that both the retinal and the optic nerve lesions should be included under the caption traumatic syndrome of the posterior pole of the eye from nonocular trauma.

A lengthy bibliography is appended.

C. E. FINLAY.

MEASUREMENT OF THE WIDTH OF THE RETINAL ARTERIOLES IN VIVO. E. LOBECK, Arch. f. Ophth. **142:** 241, 1940.

The author gives detailed directions for the use of a special measuring eye piece for the ophthalmoscope of Gullstrand (*Arch. f. Ophth.* **136:** 440, 1937). The front lens of this eye piece has been cut into an upper and a lower half which can be displaced laterally against one another by a micrometer screw. Lateral displacement of one half of the front lens entails displacement of the corresponding half of the image of the fundus. The width of a retinal arteriole is determined by turning the measuring eye piece until the line dividing the two half lenses or half images lies at right angles with the arteriole to be measured. Then one half lens is displaced until the displacement of the corresponding half of the image equals the width of the vessel. The amount of displacement is read on a drum attached to the micrometer screw. Thus a relative value for the width of the vessel is obtained which can be converted into an absolute one by measuring in the same eye with the same instrument the horizontal diameter of the disk, which the author assumes to be 1.5 mm.

P. C. KRONFELD.

Trachoma

TRACHOMA AND SULFANILAMIDE. J. L. PAVIA, Arch. de oftal. de Buenos Aires **15:** 400 (Aug.) 1940.

This paper gives an account of the work of the Argentine Committee for the Prevention of Blindness and War Against Trachoma in connection with the latter issue. The committee localized trachoma in different areas, classifying it into different types according to MacCallan.

The following dosage was used when sulfanilamide was employed: 0.04 Gm. per kilogram of body weight a day for five days, 0.03 Gm. per kilogram a day for five days and 0.02 Gm. per kilogram a day for five days.

There were no general disturbances, there was a rapid decrease in the complicating inflammations and the nodules disappeared in fifteen to twenty days, with marked reduction of the pannus.

C. E. FINLAY.

Vision

ILLUMINATION STANDARDIZATION OF THE SNELLEN CHART. H. G. MARTIN, Am. J. Ophth. **24:** 287 (March) 1941.

Martin discusses the question of standardized illumination in testing visual acuity and describes an apparatus which will give different illuminations by varying the distance of the source of light. A light meter is incorporated.

W. S. REESE.

"NIGHT-BLINDNESS": A PSYCHOLOGICAL STUDY. E. WITTKOWER, T. F. RODGER, G. I. SCOTT and B. SEMEONOFF, Brit. M. J. **2:** 571 (Oct. 25); 607 (Nov. 1) 1941.

Fifty-two unselected soldiers complaining of night blindness were examined ophthalmologically, psychiatrically and with dark adaptation

tests. The psychiatric examination showed that the great majority suffered from severe psychologic disorders and aside from their night blindness were unfit for military service because of these. The ophthalmologic examination disclosed in only 1 case evidence of a fundus lesion, resembling retinitis pigmentosa, which could cause night blindness. The dark adaptation examination showed mild disturbances of dark adaptation in 16 patients and none in 9 patients. A moderate disturbance of dark adaptation was found in 11 patients and a severe disturbance in 4. The examination was made with the Nagel adaptometer and the method of testing described by Semeonoff (*Brit. J. Psychol.* **32:** 136, 1941).

Of 34 patients studied jointly, 22 had no disturbance of dark adaptation. Most suffered from severe psychiatric disorders. Of the remaining 12 patients with definite disturbances of dark adaptation, 1 had retinitis pigmentosa and another hereditary night blindess. In practically all the others severe disturbances of ocular function like those generally regarded as "hysterical" were found. No evidence of vitamin A deficiency as an etiologic factor in night blindness could be obtained. Therefore, the belief in the value of the dark adaptation test for the discovery of night blindness and as an indication of vitamin A deficiency appears doubtful.

The authors conclude by stating that most night blindness seen in England is probably of psychologic origin. ARNOLD KNAPP.

Therapeutics

RATIONAL LOCAL AND GENERAL SYNCHRONOUS TREATMENT IN OPHTHALMOLOGY. W. LÖHLEIN, Klin. Monatsbl. f. Augenh. **104:** 533 (May) 1940.

Löhlein stresses the importance of local and general treatment in ophthalmology to be applied synergetically at the proper time and at the proper place. Extensive experiments on animals proved that the heterogenous substances brought into the blood stream (no matter whether orally, subcutaneously or intravenously) show synchronous and quantitative rules, which should be considered in ocular therapy. Many active drugs are applied into the blood circulation for the purpose of influencing directly the tissues of the eye and the pathogenic germs in them. The intended influence is inhibited by the cytotoxin retention of the secretory apparatus of the eye, a retention by means of which a number of highly molecular substances circulating in the blood stream are prevented from entering the eye. It is the so-called barrier between blood and aqueous humor, comparable to the blood-brain barrier. To get a sufficient quantity of medicaments to pass this barrier would require the application of large doses, which might damage the general organism. Experiments have shown at which time the highest concentration of the substances may be expected in the blood serum. This is the proper moment to "break the barrier" by some hyperemizing treatment of the diseased eye. The choice among the divers methods of producing hyperemia depends on the depth of the seat of the focus in the eye. The highest concentration in the blood occurs one to two hours after a subcutaneous injection, at which time production of

hyperemia would be most effective. Four hours after oral medication there occurs the highest concentration in the blood, and it will last for some time; therefore, hyperemia is best produced four to six hours after medication. The most favorable synchronous action may be obtained by cumulative oral doses. The smallest concentrations in the aqueous humor and in the vitreous were noted after intravenous administration, because with this type of administration the substance leaves the blood stream especially fast. Intravenous injections are useful in the introduction of substances which act on the interior of the eye in small doses. Some substances enter the eyeball only in small or ineffective quantities without the help of artificial hyperemia, although large quantities may have been introduced into the body. This is due to the fact that these substances when introduced into the blood stream may reach their highest concentration in a synchronous manner in aqueous humor and in vitreous but that the point of highest concentration varies according to their filtrability.

K. L. STOLL.

Society Transactions

EDITED BY DR. W. L. BENEDICT

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Symposium on Vertigo

Ocular Vertigo. DR. FRANCIS HEED ADLER, Philadelphia.

Vertigo has been defined as "the consciousness of disordered orientation of the body in space" (W. Russell Brain). The usual symptoms are dizziness, a sense of movement within the body itself, nausea or even complete prostration. In addition, certain objective disturbances, such as pallor, alterations in the pulse rate and blood pressure, sweating, vomiting and diarrhea may occur.

It can nearly always be said that severe vertigo is never of ocular origin. True ocular vertigo may arise under the following conditions:

1. *Abnormalities in the Dioptric Apparatus.*—Persons who wear glasses for the first time, especially if the lenses are of the fairly strong convex type, may complain of mild vertigo. This is due largely to the alteration in the size of the retinal images when the lenses are not placed exactly at the anterior principal focus of the eye.

2. *Paralysis of Extraocular Muscles.*—The most severe form of ocular vertigo is that which arises at the start of paralysis of an ocular muscle. This is due to two factors: diplopia and a disagreement between the visual sensations and those of other spatial localizations.

3. *Optokinetic Nystagmus.*—Persons subject to car sickness have optokinetic nystagmus and probably vertigo. In some cases correction of a heterophoria by prisms affords relief from nausea when the patient is traveling by train or by automobile. Many vertical hyperphorias are due to a paretic muscle, and it may be that during optokinetic nystagmus the hyperphoria becomes hypertropia.

4. *Looking Down From Heights.*—The largest factor in the production of this type of ocular vertigo is lack of the customary vanishing point. Ocular vertigo due to heights disappears completely if one is in a plane, particularly if it is possible to look over the side out of the cockpit and get a view which does not include part of the plane in the visual field.

5. *Effects of Accelerations of the Body.*—Armstrong concluded that vertigo when produced by accelerations occurs only when conflicting sensory impressions from two or more of the organs of equilibrium reach consciousness. This factor becomes of extreme importance in

blind flying. As long as a pilot is able to have a visual reference to the ground he will not suffer from vertigo due to accelerations unless these are very violent.

Vertigo from the Standpoint of the Otolaryngologist. Dr. W. J. McNally and Dr. E. A. Stuart (by invitation), Montreal, Canada.

Dizziness that has come on during movement, particularly in a person who has cardiovascular disease or some other abnormality of the circulation to his labyrinth, may be the result of an abnormal labyrinthine reaction. In Ménière's syndrome there is some type of abnormal stimulus. Irritation of the labyrinth will cause extremely severe dizziness, and the classic description is that it is sudden and rotary. When dizziness is so described, it usually means labyrinthine stimulation. As a matter of fact it is not diagnostic. Reports of 31 cases of angle tumor were reviewed to determine this point. There was a history of dizziness in 17 of them. Six of the patients had varying degrees of sudden rotary dizziness. In 1 case, for two years before any signs diagnostic of intracranial disease appeared the patient had sudden attacks of rotary dizziness, deafness and tinnitus, which symptoms are diagnostic of Ménière's syndrome. He finally had well marked symptoms of intracranial disease and was operated on for a tumor of the eighth nerve. Of our series of cases in which the diagnosis was Ménière's syndrome, there was sudden rotary nystagmus in only 50 per cent. Of 50 cases of hypertension and cardiovascular disease there was a history of dizziness in 21, of 23 cases of hypotension there was a history of dizziness in 10, of 90 cases of disease of the gallbladder there were complaints of dizziness in 18 and of 50 cases of diabetes there were complaints of dizziness in 8.

We concluded that in analyzing an attack of dizziness one should find out if the dizziness occurred during movement—the result of an abnormally reacting labyrinth—or whether it occurred while the patient was at rest—the result of an abnormal stimulus. In cases of true Ménière syndrome it is more likely to be of the latter type. Labyrinthine dizziness is not necessarily rotary in character. Dizziness in the course of chronic suppuration of the ear most likely indicates inadequate drainage of the middle ear.

Vertigo: Its Neurologic Features. Dr. Bernard J. Alpers, Philadelphia.

Vertigo is a symptom of disease of the peripheral vestibular mechanism or its central pathways. These pathways are in intimate association with the vestibular labyrinth, the brain stem, the cerebellum, the ocular muscles and the cerebral cortex, as a result of which impressions received from many parts of the body are synthesized for the maintenance of equilibrium. When disease affects one of the many sectors within the brain which are concerned with transmission of vestibular impulses, vertigo results.

From a diagnostic standpoint, the problem of vertigo has two aspects: first, the problem of localization, whether it is of peripheral origin or of central origin, and second, the determination of the precise

central localization once it is determined that the vertigo is not of peripheral origin. The problem of differentiation of vertigo due to labyrinthine disease from that of central origin is of great significance. The main distinguishing features are these:

1. The episodic character of the attack.
2. The nature of the vertigo.
3. The occurrence of nystagmus.
4. The presence of associated signs of central involvement.
5. The results of the Bárány test.
6. The presence of cochlear signs.
7. The presence of vegetative symptoms.

If the vertigo is of central origin, the chances are that the causative process lies in the posterior fossa, particularly within the brain stem and probably in the medulla, but is less likely to be found within the pons or the midbrain. Next in order of probability is the cerebrum, but occurrence of the causative process is far less likely in this region than in the brain stem. The next less likely region is the cerebellopontile angle, and the least likely region is the cerebrum.

Of the various degenerative lesions which may involve the brain, multiple sclerosis is an important cause of vertigo. Vertigo may be the first and for some time the only symptom of this disease, and it is the result of involvement of the brain stem. Vertigo in association with multiple sclerosis is often transitory, but it may be persistent. Vertigo may occur after any acute injury to the head. It is most common after concussion of the brain with which fracture of the skull or damage to the brain is not associated.

Surgical Treatment of Vascular Diseases Altering the Functions of the Eye. Dr. Alfred W. Adson, Rochester, Minn. (by invitation).

The lesions discussed are those that alter the position of the globe, interfere with normal movements of the eye and impair the fields of visual acuity. They include such lesions as arteriovenous fistulas of traumatic origin which develop between the internal carotid artery and the cavernous sinus, varicosities of the orbital veins, vascular tumors of the orbit, aneurysms of the middle cerebral artery, aneurysms of the arteries composing the circle of Willis and fusiform enlargement of the internal carotid artery which produces pressure on and distortion of the optic nerve.

The surgical procedures employed vary according to the lesions encountered. In some instances attempts are made to trap the aneurysm or fistula by ligating the vessel on each side of the lesion. In other instances such operations are employed as venectomy, decompression of the optic nerve by liberating or unroofing of the nerve by rongeuring away bone from the optic canal or by removal of bone from the roof of the orbit and removal of tumors of vascular origin.

DISCUSSION

Dr. Wendell L. Hughes, Hempstead, N. Y.: Dr. Adson has ably discussed vascular lesions affecting the visual function from the neuro-

surgical standpoint. He has covered the surgical treatment of arteriovenous aneurysms so thoroughly that little remains to be said. Most of these lesions are out of the field of the ophthalmic surgeon. I therefore have had no personal experience with the method he advocates.

The procedure is certainly one that has been carefully developed surgically and is based on a detailed study of the normal and the pathologic anatomy and physiology involved. The fact that the vision of the ipsilateral eye was not affected in the cases in which the ophthalmic artery was ligated shows that there must have developed some blood supply to the retina through its central artery other than that which came from the internal carotid through the ophthalmic artery. That there was definite establishment of a flow of blood was shown in case 3, in which the pulsating flow of blood into the internal carotid from the ophthalmic artery in a reverse direction was demonstrated before the latter was ligated. It ceased when the artery was ligated.

Dr. Adson mentioned that in 1 case the exophthalmometer reading was 18 mm. for one eye and 34 mm. for the other. With this amount of exophthalmos the vitality of the cornea might be endangered. I should like to ask Dr. Adson if he found it necessary to use lid adhesions to prevent loss of the eye from exposure of the cornea. This procedure might become necessary in cases of extreme exophthalmos. I wonder if Dr. Adson has ever had to resort to stuffing the cavity of an aneurysm with muscle tissue when the aneurysm could not be tied off, as in a case reported by Dr. Jefferson Browder.

Ophthalmologists are often called on to treat hemangioma affecting the lids, the eye or the orbit. The surgical treatment of the small tumor of this type presents little difficulty, but the large tumor affecting the deeper structures is often more difficult to attack satisfactorily. Dr. Adson in his discussion of these lesions did not mention the use of the cutting and the coagulating diathermy current. In the removal of some of these growths the bleeding is so profuse that thorough removal becomes almost impossible unless the bleeding can be kept under control. Much of the bleeding can be controlled by making incisions with the cutting current and with the coagulating current touching bleeding points and destroying veins that cannot be excised.

Among the vascular conditions affecting the function of the eye must be included various conditions affecting the blood vessels of the orbit and the vessels of the eye itself.

Dr. Wheeler and others have reported cases of pseudotumor consisting of a cyst without epithelial or endothelial lining filled with blood in various stages of decomposition. In these cases the history was of only minor traumatism, apparently irrelevant. The patient recovered when the contents of the cyst were released and the sac obliterated by sutures. The diagnosis in such cases is difficult, but aspiration will usually settle the problem.

Surgical treatment of sudden closure of the central artery of the retina has in my experience been most disappointing. Paracentesis of the anterior chamber in the great majority of cases produces no results. Occasionally there will be some spontaneous recovery of function, especially if only a branch is involved. The same prognosis applies to expulsive hemorrhage of the choroid, which occurs in about 1.5 per cent

of cases of cataract extraction. Incision of the sclera has been tried in these cases with some success.

I think in general it should be remembered by ophthalmic surgeons that tumors deep in the upper part of the orbit, especially if situated nasally, are much more satisfactorily approached transcranially by the neurologic surgeon, as Dr. Adson has pointed out, with a better chance for investigation and total removal if necessary than from in front by the ophthalmic surgeon. The neurologic surgeon's training fits him to take care to better advantage of unforeseen conditions that may be encountered. If the lesion requires exploration of the lower, or temporal, part of the orbit, the operation is definitely in the field of the ophthalmic surgeon, and the approach may be either through a frontal incision or by the Krönlein method.

Referring to Dr. Adson's slides showing bilateral fusiform enlargement of the ophthalmic artery pressing against the optic nerve, I should like to ask him if there was any change in the shape of the optic foramen seen by roentgenogram preoperatively. I judge from his remarks, however, that the lesion was not suspected preoperatively and that a roentgenogram of the optic foramen may not have been made

Dr. Alfred W. Adson, Rochester, Minn.: I am grateful to Dr. Hughes for his discussion. In reply to his inquiry concerning the necessity to use lid adhesions to prevent loss of the eye from exposure of the cornea with severe proptosis, I would state that after ligation of the internal carotid and the ophthalmic artery and removal of the veins from within the orbit the proptosis disappeared and the lids protected the eye. At the conclusion of the operation I introduced sterile petrolatum between the lids and applied petrolatum over the lids before placing over the orbit a gauze pack which was held in place with a roller bandage. This extra precaution was employed to maintain the position of the eye during the period of postoperative edema.

In reply to the second question, concerning the use of muscle tissue with the hope of closing the arteriovenous fistula, I can answer in the affirmative. I have used the Brooks technic, which consists of exposing the carotid arteries in the neck, opening the internal carotid and stuffing bits of muscle into the artery with the hope of filling the artery and plugging the cavity of the aneurysm. The procedure has not been too satisfactory, since the bruit and the proptosis did not completely disappear. In order to determine the effectiveness of the muscle-stuffing procedure, I applied a silver clip to the first strip of muscle introduced into the carotid artery in order that I might identify it on the roentgenogram. The film did show that the clip was situated in the proper position, but apparently the muscle did not encourage sufficient thrombosis to close the arteriovenous fistula that existed between the internal carotid artery and the cavernous sinus.

I did not go into a discussion of the surgical management of angiomatous tumors within the orbit, since the technic is a fairly standardized procedure. I invariably employ electrocoagulation to prevent bleeding from the small arteries and veins, using caution not to overheat the structures in the vicinity and thus impair the function of the oculomotor nerve. I am unable to answer offhand the question as to whether the optic foramens in the 4 cases of arteriovenous fistula reported were enlarged,

but I have roentgenograms of the optic foramens made in all cases of neurosurgical lesions in and about the orbit. I am inclined to believe that roentgenograms were made and that the optic foramens were not enlarged, for I attempted in preparing the abstracts of the histories to record all abnormal findings. In 2 cases there was definite evidence of bony erosion, which had resulted in a thinning of the dorsal orbital plate. I have also seen and operated on a number of patients with meningioma situated in the optic foramen who presented a markedly enlarged optic foramen.

Intermittent Exophthalmos (Postural Exophthalmos); Report of a Case. Dr. W. A. Poole, Lexington, Ky.

Any protrusion of the eyeball which is not constant is classified as intermittent exophthalmos in this discussion. Special attention is given to those cases in which the condition is caused or accompanied by varicosities of the ophthalmic vessels. Exophthalmos was the most important symptom in the case reported.

Exophthalmos is described as being provoked by certain postures. The proptosis is usually of short duration. Occasionally the protrusion occurs spontaneously, without apparent cause. Early subjective ocular symptoms are frequently absent, the exophthalmos being noted by another person. Rarely is pain a prominent symptom. Severe disturbances of vision are uncommon. Dizziness, vertigo and tinnitus frequently cause the patient to consult an otologist.

If the patient should be examined in a sitting position, no clinical pathologic process will be found and the true condition may be overlooked. Having the patient bend forward might well be made a part of the routine ophthalmic examination, especially when there is a history of dizziness or of a sensation of fulness in the head.

The etiologic factor is not certain. It varies with the different types of exophthalmos. Varicosity, vascular tumor and aneurysm are the most common causes. The pathologic process also is uncertain. Patients who have suffered from intermittent exophthalmos rarely have come to necropsy.

Compression of the carotid artery, injection of solutions which cause sclerosis of the vessels, radium therapy, roentgen ray therapy and surgical intervention are some of the methods suggested for treatment.

DISCUSSION

Dr. Edwin B. Dunphy, Boston: Dr. Poole's paper deals with an interesting and dramatic condition and suggests that its frequency may be much higher than most ophthalmologists have been led to believe. I have come across only 1 case of the classic type of intermittent exophthalmos, in which it was due presumably to varices in the orbit. In my case the past history and the family history were irrelevant and the physical examination revealed no abnormalities. The patient was a young man in his twenties whom I had had under observation for the past six years. At the age of 18 he first noticed that whenever he did any physical exercise requiring forced holding of his breath, such as chinning the bar or lifting a heavy object, he felt as if his left eye was going to come out.

Examination of the affected eye, which was the left one, revealed complete absence of exophthalmos in the position of rest; in fact, slight enophthalmos was present. On forcible holding of the breath, however, this eye protruded as much as 8 mm. as measured on the exophthalmometer, and it slowly returned to its normal position on resumption of normal respiration. During the period of protrusion there was no evidence whatever of dilatation of any veins, externally or ophthalmoscopically. As the patient was much annoyed by the condition, I attempted to correct it by injecting sclerosing solutions in the orbit, as advocated by Weyman and others.

In 1925 Reverdino reported a complete cure of the condition by injection of a sclerosing solution, and Dr. Poole mentioned Weeker's case, which was reported in 1933 and in which he also recorded relief by this method of treatment.

In my case I injected 0.3 cc. of half-strength quinine hydrochloride and ethyl carbamate (urethane) solution on three occasions without any appreciable effect on the condition.

I should like to ask Dr. Poole how he explains the persistent closure of the lid during the episodes of protrusion in his case. Perhaps it was a protective measure on the patient's part in order to hold back the ocular spasm, which he might have felt coming on. In my case no such spasm of the lid occurred. There was out-and-out proptosis of the eyeball.

It is interesting to speculate on the mechanism of this protrusion, and I think all one can do is to speculate, in the absence of any adequate pathologic material. The basic fact to be kept in mind is that the condition develops because of an overfilling of varicose orbital veins under certain conditions which increase venous pressure. It is known that blood is drained from the orbit by three ways: first, backward by way of the ophthalmic veins into the cavernous sinus (most ophthalmologists feel that this is the main course of exit of blood from the orbit); second, forward into the facial system by means of communications between the ophthalmic veins and the angular veins, and third, downward through the inferior orbital fissure into the pterygoid plexus.

Apparently, then, the blood in the orbit can escape by these three different routes, and the position of the head may possibly affect in some measure the method of escape. Some anatomists have demonstrated several points in the courses of these various orbital channels where compression might occur and cause obstruction to venous return from the orbit, but the free communications between the ophthalmic veins and all the orbital veins and the fact that the orbital veins have no valves would seem to compensate for any theoretic obstruction that might occur along the course.

Whitnall has called attention to the fact that the channel formed by the ophthalmic veins as they empty into the cavernous sinus is considerably smaller than one would expect in view of the number of venous radicles which it collects. Furthermore, he pointed out that the cavernous sinus is filled with the internal carotid artery and also a considerable amount of trabecular tissue and, therefore, one might expect sluggishness of circulation.

Of course, these facts alone are not enough to explain intermittent exophthalmos, but when the exophthalmos is accompanied by some other

possible impediment to the flow of the blood, either downward into the pterygoid plexus or forward into the facial system, it may be enough to explain why a sudden increase of venous pressure in the orbital veins gives rise to an enlargement of the preformed varix in the orbit, with consequent protrusion of the eyeball.

Dr. Poole has called attention to anatomic factors. I believe he quoted Reese as saying that the left jugular foramen as it leaves the skull is usually smaller than the right. This might be responsible for the fact that when varix does form in the orbit it forms on the left side, and, as Dr. Poole has stated, the incidence of this condition is ten times more frequent on the left than it is on the right.

To sum up the situation, I might say that in the present state of knowledge, I believe one can do no more than agree with Birch Hirschfeld about this condition, and he said: "Intermittent exophthalmos depends on a varix of the orbital veins, probably congenital in origin, although usually the venostasis does not take place until much later on in life, and this occurs under the influence of possible mechanical obstructions to blood flow which are merely presumptive on our part and to which we have already alluded."

Dr. Ralph O. Rychener, Memphis, Tenn.: I wish to report a case of intermittent exophthalmos in a patient 58 years old. The reason for presenting this particular case is that there has been some improvement as time has passed. Dr. Ellett reported it before the American Medical Association in 1940 (Ellett, E. C.: Unilateral Exophthalmos, Tr. Sect. Ophth., A. M. A., 1940, p. 50). Dr. T. C. Chapman, of Brownsville, Tenn., had reported it in 1931 before the Memphis Society of Ophthalmology and Otolaryngology.

For more than twenty years the left eye has protruded 2 to 10 mm. whenever the patient has had a cold, has worn a tight collar, has leaned over forward or has happened to fall asleep on his face. The protrusion has been accompanied by a severe aching pain behind the eye. Pressure on the right jugular vein has had no effect, but pressure on the left jugular vein has caused the protrusion, which has been associated with congestion of the conjunctival vessels and enlargement of the retinal veins of both eyes. The condition is probably due to a vascular tumor in the left orbit which is of negligible size unless filled with blood because of some obstruction to the venous flow of blood from the head.

With the passage of time the tendency toward exophthalmos has decreased, and now only on certain occasions is the patient able to demonstrate the condition. Last summer motion pictures were made of him attempting to produce exophthalmos by lowering his head between his knees and by pressure on the left jugular vein. However, on the day they were taken the patient knew that the protrusion was not pronounced.

Although the exophthalmos is much less marked now than it was on the occasion of the original demonstration before the Memphis group, a protrusion of 2 mm. can be produced by pressure on the left jugular vein.

Dr. Benjamin Rones, Washington, D. C.: I would have preferred to hear this discussion last week, because it happens that my first patient with this condition, a woman of 41, was seen last Saturday. Her left eye also was involved. She gave absolutely no history of injury. The

story is that nine years ago there were some shooting pains in the eye, and the patient discovered that whenever she stooped or slept on her left side—not on the right side, as in Dr. Poole's case—she would have unilateral exophthalmos.

When she told me this story, I was somewhat skeptical, and when she bent over and came up again it was a bit of a shock to see a protrusion of 3 to 4 mm.

Like Dr. Dunphy's patient and unlike the essayist's, there were no congestive signs whatever. The patient simply had exophthalmos, without, however, any drooping of the lid. There were widening of the palpebral fissure and a definite lid lag when the patient looked downward, with some weakness on convergence. The screen test showed interference with the movements of the eye, and when the diplopia fields were taken it became evident that she had paresis of the superior oblique muscle of the left eye.

I offer no theory as to causation, but simply report a phenomenon that is striking when first seen.

Dr. W. A. Poole, Lexington, Ky.: I wish to thank the gentlemen for their discussions.

Dr. Dunphy mentioned closure of the patient's eyelids as the proptosis increases. The lids become thickened and hard, and it is difficult to separate them.

The patient has no perceptible photophobia.

I was glad to hear of Dr. Rones's case. His discovery of the phenomenon increases my belief that forward bending of the head should be a part of routine ophthalmic examinations. It will be interesting to hear more about his case.

Myopia. Dr. Alfred Cowan, Philadelphia.

In the minds of a great many ophthalmologists there still exists the belief that every myopic eye is potentially a diseased eye. On the basis that any form of myopia is a disease, theory after theory has been proposed to explain its incidence and its progress. Not one of these theories has ever been proved clinically; probably every one can be shown to have no scientific evidence and many are ridiculous from a scientific standpoint. Simple myopia is nothing more than a refractive error, depending generally on an increase in the length of the eyeball over what is accepted as normal. It is no more a disease than hypermetropia or astigmatism. No simple refractive error, whether it is a myopia, a hypermetropia or an astigmatism, is a disease.

In the light of present knowledge neither myopia nor any other refractive error can be produced or controlled any more than can the separate growth of any other feature of the body. The management of simple myopia consists of merely providing the proper corrective lenses. The main objective is to provide normal visual acuity and comfort. No medical treatment or hygienic measures have ever been shown conclusively to have any effect one way or the other.

Use of the term malignant myopia should be discontinued. So-called malignant myopia is nothing more than a disease of the eye, usually

some form of uveitis. It may be associated with myopia, or myopia may be the outcome. The eyes affected are really diseased eyes, which may eventually go on to blindness. Surely blindness has never been caused by a refractive error.

DISCUSSION

DR. E. C. ELLETT, Memphis, Tenn.: It has long been known that the eyes of an infant are hyperopic and that the hyperopia tends to decrease as the child grows; usually hyperopia persists in some degree, though it may continue to decrease until the child is grown and may pass over into myopia. This tendency is recognized in the recent publications from the United States Naval Academy to the effect that a student who has 1 D. of hyperopia or less is apt to become myopic by the time of his graduation and will not be suitable material for a naval officer.

The idea formerly prevailed that all myopia tended to progress indefinitely and end in disaster, but it is known now that there is a limit to the amount of increase and that the usual type, which Parsons called "developmental myopia," runs about the following course:

A child who has had no previous complaints and who possibly on several occasions has been found to have normal vision begins to complain at about the age of 8 or 10 of poor distance vision (blackboard, movies, etc.) and is found to have myopia of 1 D. or so. In spite of everything that is done, or if nothing is done, the myopia increases until the age of about 20, to the extent of 3 or 5 D. and then becomes stationary. In answer to the distress of the parents over the child's wearing glasses, especially if the patient is a girl, everything possible is tried, including glasses, to arrest the progress and has not the slightest effect. This does not mean that in all cases the myopia progresses, for in many it does not, and in many it progresses only to 2 or 3 D. and then stops.

Treatment.—Of the many measures advised to alter the course of the disease, if it may be called that, the following may be mentioned: (1) the prescribing of glasses, (2) the restricting or prohibiting of near work, (3) the use of epinephrine, (4) the atropinization of one eye and (5) general treatment.

In attempting to evaluate these measures I have analyzed the records of 300 private patients, not selected but taken as they came, who have been under observation from three or four to forty years.

1. Prescribing of Glasses: Most textbooks advise giving a full correction, or —0.25 D. sph. more than the full correction, for constant wear. This is helpful as far as the vision is concerned, but my experience is that the correction has no effect in preventing the increase of the myopia. I can report that under this plan some patients did not show any progress in their myopia, but an equal number who wore glasses occasionally showed the same tendency to remain stationary. A few typical cases are reported in the accompanying tables.

If the patient is a girl she and her mother are sure to cooperate to the end that she leaves the glasses off on social occasions, to which I see no objection.

I have under observation a few women whom I have known for forty years or more and who have worn their glasses little or almost

not at all, and these women, of 60 or past, are no worse off than others with the same type of trouble who have worn their glasses constantly. In the wearing of glasses, as in other plans for treating myopia, one is

TABLE 1.—*Cases Illustrating Typical Developmental Myopia*

	Age at First Test, Yr.	Error at First Test	Age at Which Myopia Began, Yr.	Error After 2 Years	Error After 5 Years	Error After 10 Years	Error After 20 Years
1.....	8	+ 0.50 D. sph.	12	— 2.00 D. sph.	— 2.25 D. sph.	— 3.00 D. sph.	
			(— 1.50 D. sph.)	— 1.50 D. sph.	— 1.75 D. sph.	— 2.75 D. sph.	
2.....	10	— 3.50 D. sph.	8	— 4.00 D. sph.	— 5.00 D. sph.	Same	Same
				— 4.50 D. sph.	— 5.50 D. sph.		
3.....	9	Emmetropia	16	..	— 1.75 D. sph.	— 2.25 D. sph.	No change after 19
					— 0.50 D. sph.	— 2.50 D. sph.	

TABLE 2.—*Cases Illustrating Course of Myopia over Many Years*

	Age at First Test, Yr.	Error at First Test	Age at Which Myopia Began, Yr.	Error After 5 Years	Error After 30 Years	Error After 45 Years
1.....	16	— 4.50 D. sph., both eyes	14	— 4.75 D. sph., both eyes	— 5.00 D. sph.	
					— 4.00 D. sph.	
2.....	7	— 4.00 D. sph., both eyes	7	— 7.00 D. sph.		— 8.50 D. sph.
				— 8.00 D. sph.		— 9.00 D. sph.

TABLE 3.—*Cases in Which Epinephrine Was Used for Two Years*

	Age at First Test, Yr.	Error at First Test	Age at Which Myopia Began, Yr.	Error After 2 Years	Error After 5 Years	Error After 10 Years
1.....	6	— 0.50 D. sph.	6	— 1.00 D. sph.	— 1.75 D. sph.	— 3.25 D. sph.
				— 0.75 D. sph.	— 1.25 D. sph.	— 3.00 D. sph.
2.....	14	Emmetropia	16	— 0.50 D. sph. and cyl.	— 1.00 D. sph. and cyl.	— 1.00 D. sph. and cyl.
3.....	8	— 1.50 D. sph.	7	— 3.00 D. sph.	— 4.25 D. sph.	
					— 3.50 D. sph.	
4.....	16	— 1.25 D. sph.	15	— 2.75 D. sph.	— 4.25 D. sph.	
		— 1.50 D. sph.		— 3.00 D. sph.	— 3.75 D. sph.	

struck with the analogy to one's experience in treating cataracts. Some cataracts progress and some do not, and it is difficult to say whether treatment has any effect in either condition.

The question of the value of contact glasses in treating and preventing the progress of myopia is still undecided. My feeling is that since such glasses do not and should not make pressure in the eye, they could not do any good. I hesitate to advise them until the myopia is stationary.

2. Restricting or Prohibiting of Near Work: It is a common observation that myopic children are bookworms and find their main source of entertainment in reading. It is impossible to suspend accommodative acts effectively, because at the table, in conversation, in brushing the hair, etc., the child depends on accommodation. If a child is taken out of school, how long must he stay out? What assurance can be given that the myopia will be arrested by taking him out? I have met this phase of the situation by advising that the eye work be limited to necessary school work and the child be encouraged to entertain himself by other means—such as swimming, skating, dancing and outdoor sports —but such advice is not often strictly followed, nor do I know that it helps if it is.

3. The Use of Epinephrine: Meyer Weiner has advised the use of 1:1,000 epinephrine hydrochloride three times a day as a means of preventing the progress of myopia, and Gradle and others have reported favorably on this method. Of my 300 patients, epinephrine hydrochloride was prescribed for 103 children. It was not used faithfully by many of them, and I would consider that its use for less than a year is not a fair test; however, among the patients who tried it that long I have not observed more than an occasional cessation of the increase in the myopia. I can report quite as many who received no treatment or only wore glasses and followed a hygienic regimen in whom the myopia did not progress. My experience with epinephrine, therefore, is not encouraging.

4. Atropinization of One Eye: This plan was suggested and the details explained by W. H. Luedde (Monocular Cycloplegia for Control of Myopia, *Am. J. Ophth.* **15:** 603 [July] 1932). It harks back to an observation by Donders to the effect that watch makers, who use a monocular loupe, do not show progress in myopia. The suspension of the accommodation in one eye by atropine abolishes or diminishes the effect of convergence, which many observers think is the potent factor in progressive myopia. It may explain some cases, and in these the method is worth trying. Luedde reported favorably on it. I have suggested it to several patients, but so far have not been able to give it a fair trial. Convergence does not explain the myopia in all cases, nor did Luedde claim that it does. For example, a boy of 10 was seen in May 1941, having lost the vision in the left eye in September 1940 as the result of an accident. He was seen at the time of the injury by Dr. Donald O'Rourke, of Denver, who did not measure the refraction of the right eye but noted vision of 6/6, which certainly excluded myopia at that time. In May 1941 a test with homatropine showed vision of 6/15 and myopia of 1 D.

5. General Treatment: Such general tonic and other treatment as is indicated should be given, and some children need certain vitamins, especially vitamin A, but there is nothing specific in their use as far as myopia and its progress are concerned.

Comment.—The myopia that is congenital or that becomes high at an early age is quite a different matter and is not, I take it, under con-

sideration. I have recently seen a child less than a year old with myopia of between 20 and 30 D. as measured with the ophthalmoscope, and others with myopia of less, though almost as high, degree.

My experience and the opinions resulting from it agree closely with Dr. Cowan's. It does seem to me, however, that myopes are particularly susceptible to low grade uveitis, of which the commonest manifestation is opacities of the vitreous, but I am not sure which is the cart and which is the horse. They are also, as a group, subject to more and greater changes in their refraction than other persons are, and for this peculiarity I do not know a satisfactory explanation.

DR. ARTHUR ALEXANDER KNAPP, New York: Dr. Cowan's remarks are interesting, but it seems that there are varying results, particularly with vitamin therapy. Dr. Cowan stated that myopia is a localized condition. It is known that myopia of any type is not purely a localized refractive error but has a systemic status, just as there is a tuberculous status. This was recognized years ago.

In the résumé of his paper Dr. Cowan stated that in the light of present knowledge neither myopia nor any other refractive error can be controlled any more than separate growth of any other feature of the body can be controlled. It might be said that after the administration of viosterol and mineral mixture tablets not only the eye but the whole body may undergo a change.

Briefly, I should like to outline the work that has been done in the Department of Pharmacology at Columbia University. Blackberg and I have carried on experimental work with dogs and rats, inducing what we felt was myopia due to a diet deficient in vitamin D and calcium. We enucleated some eyes, and under the microscope the pathologic process observed was primarily a generalized edema of the fibrous tunic, which caused a weakening of this outer coat. Subsequently, at the New York Eye and Ear Infirmary, I carried out research work on patients with anterior curvature and progressive axial myopia. Careful examinations of their eyes revealed less axial myopia in some and improvement in all patients with keratoconus. Accurate measurements of plaster of paris casts of the eyes were made by Prof. Carlos de Zafra, of the department of engineering, and Prof. Louis Granath, of the department of physics, both of New York University. They carefully measured the casts, and their findings corroborated my clinical experience. Definitely they showed that the eyeball may actually shrink after the administration of vitamin D and calcium. More than seventy casts representing both types of myopia have been measured, and the improvement is macroscopic.

Furthermore, as to progressive myopia, over 50 per cent of the patients showed either that they had less myopia or that it remained stationary after vitamin D and calcium therapy. Supplementary reports, unpublished, corroborate these findings.

I should also like to show that the fundus of the eye, or the posterior segment, may shrink. This fundus picture (slide) is that of a woman of 39 who had myopia of 12 D. and after the administration of vitamin D and calcium showed a shrinkage of the posterior segment of the eye. The photographs were taken under identical conditions at the New York Eye and Ear Infirmary by the same photographer. It may

be stated that the measurements were made by Mr. Host, of the American Optical Company.

This view (slide) is the first taken before therapy. The vertical distance from this fixed point to this (the superior and the inferior margin of the central staphyloma) was measured at 3 feet 1 inch (94 cm.). These fixed points (the crossing of the vessels at the superior border of the disk and the crossing of the vessels in the macular area) being used, the horizontal distance was 4 feet 2½ inches (128 cm.).

This view (slide) was taken after six months' treatment with vitamin D and calcium. The vertical distance between the two points now measured 2 feet 11½ inches (90 cm.), whereas the horizontal distance measured 4 feet ½ inch (123 cm.), a decrease in the vertical diameter of 1½ inches (3.8 cm.) on the screen and in the horizontal diameter of 2 inches (6.1 cm.), showing that both the vertical and the horizontal diameter of the posterior segment of the globe may shrink after treatment with vitamin D and calcium.

It should be borne in mind that no matter what classification one arbitrarily makes, it is not reasonable to expect any type of untreated myopia to be reduced. From the casts presented and the fundus photograph projected on the screen, it may be concluded that myopia actually may be reduced by a shrinkage of both the anterior and the posterior segment of the globe after vitamin D and calcium therapy.

DR. SANFORD R. GIFFORD, Chicago: I want to congratulate Dr. Cowan for his taking this courageous and unpopular position. Everybody knows that parents come to ophthalmologists with children who have something the matter with them, and want the ophthalmologists to do something for these children. They want a cure for the condition, and any one who offers to do anything for myopia at once puts himself in a position of tremendous advantage over his colleagues in the same community. I think the only thing that honest ophthalmologists can do, however, is to face the facts and tell the parents what the facts are about myopia. I think that Dr. Cowan has told essentially the whole truth in the matter.

One might speak of the opacities in the vitreous or of some of the degenerative changes of the fundus in myopia, but one knows that these also occur as senile changes, as Vogt has shown. I have taken somewhat the same position in the chapter on refraction in my textbook, and I should like to put my neck out just a little farther and take up the question of sight-saving classes. The treatment of children suffering from simple myopia by placing them in sight-saving classes seems to me to be a total mistake and to disregard entirely the real purpose of these classes, which is to afford a place for children who cannot see well to study under conditions in which they can see. It seems to me that there is no sense in putting myopic children in sight-saving classes.

There is just one suggestion as to the management of myopes that I might make: It has often seemed advisable to give them less than their full correction for reading. They often complain of asthenopia when wearing their full correction

There is one question which I should like to ask Dr. Cowan. I tell the parents that the only thing they can do for their children is to have them lead healthy lives. If they need vitamins, give them

vitamins. If they have rickets, give them vitamin D. Nobody has ever proved, it seems to me, that in a large number of myopes there is a deficiency of vitamin D or of any other vitamin any more than there is this deficiency in the average population. But I always tell the parents that they can expect the progress of myopia to stop at about the time the child reaches his full growth. Occasionally the progress seems to continue for some time past the age of 21. I should like to know what Dr. Cowan's experience has been in this respect.

DR. ALFRED COWAN, Philadelphia: The remarks of Dr. Ellett and to a great extent those of Dr. Knapp, back me up in my contention. There is no doubt that the ophthalmologist finds a number of myopic patients with opacities of the vitreous and other lesions; but he knows that such opacities, whether he sees any other lesion in the eye or not, should be considered an indication of some disease condition, past or present. There is not myopia per se, but a disease associated with myopia. One certainly sees opacities of the vitreous associated with other forms of ametropia. When Dr. Knapp mentioned anterior curvature myopia I presume that he meant keratoconus. That is not what I consider simple myopia. It is keratoconus. He said that in 50 per cent of cases myopia will remain stationary with his method of treatment. Now I do not say that his method of treatment is not good. I have not carried it out to the extent he has, and, therefore, he is in a better position than I to make positive statements; but I do not consider 50 per cent of arrests such a great result. Probably 50 per cent of the myopia in general practice remains stationary without any treatment at all.

The eye that Dr. Knapp showed on the slide is, of course, a diseased one—there is choroiditis. Treatment of such a condition has nothing to do with the treatment of myopia.

Dr. Gifford was my nicest discussant. His was the kind of discussion I like. I am glad he brought up the subject of sight-saving classes. I have been fighting them for years. I feel that this term should be dropped and the term "low vision class" substituted, since the classes do not save sight. Children who cannot be made to see well should be placed in low vision classes, and, for them, such classes are useful. I know for a fact that in Philadelphia in the so-called sight-saving classes there have been children whose visual acuity with glasses has been normal but who have happened to have myopia of more than 5 or 6 D. or whose ophthalmologists have had the idea that they must not use their eyes for ordinary work. They are placed with children having low vision, and so children who are not handicapped at all are given a certain complex. Because the ophthalmologist feels that the child might lose his sight some day, he forbids the use of the child's eyes. This is sight saving. What is he saving the child's sight for? Why shouldn't a person use his sight while he has it?

Some myopic children have even been admitted to institutions for the blind. The object is to teach them Braille or the ways of a blind person because they may some day become blind. It would be much better to educate such children to the greatest extent possible while they still see.

I explain to the parents of a child with simple myopia that the child has myopia and that myopia is nothing but a refractive error. The child

will always need glasses; but most people wear glasses nowadays and a person's eyes are just as good as they can be made to see with glasses. I emphatically state to the parents that their child's eyes are not diseased.

I think that parents should not believe nor should they lead the child to believe that because he needs concave lenses he is afflicted.

As far as the progress of the condition is concerned, it increases, sometimes up to the age of 25, 30 or even 35; but just as long as there is no disease the increase does not matter.

Operative Results in Two Hundred and Eleven Cases of Convergent Strabismus. Dr. John H. Dunnington and Dr. Maynard Wheeler, New York.

This article will be published in full, with discussion, in a later issue of the Archives.

Allergies in Ophthalmology. Dr. Albert N. Lemoine, Kansas City, Mo.

This article will be published in full, with discussion, in a later issue of the Archives.

Five Hundred Cases of Melanoma of the Choroid and Ciliary Body Followed Five Years and Longer. Col. George R. Callender, Medical Corps, United States Army; Helenor Campbell Wilder, and Col. J. E. Ash, Medical Corps, United States Army, Washington, D. C. (by invitation).

There are 1,600 cases of malignant intraocular melanoma in the American Registry of Ophthalmic Pathology, maintained at the Army Medical Museum under the auspices of the American Academy of Ophthalmology and Otolaryngology. Among the patients represented there was a slight and insignificant preponderance of males over females. Only 8 were Negroes. The tumor occurs during adult life and appears but rarely in the earlier decades. In actual numbers it occurs most often in the sixth decade, but in proportion to population the incidence appears highest in the sixth and seventh decades.

Five hundred cases of melanoma of the choroid and ciliary body have been followed five years or longer, or the patients have died as a result of metastasis before the expiration of that time. The tumors have been classified by cell type and by argyrophil fiber content. Tables are presented which show the results of the classification with relation to prognosis. The following corollaries are drawn: The pure spindle cell types, particularly subtype A, are of a relatively low degree of malignancy, and an increased malignancy is associated with the presence of epithelioid cells; a low degree of malignancy is usually associated with a heavy fiber content.

Although there appears to be an increased lethality with increased pigment content, it is felt that there is not enough variation in death percentages to justify conclusions until a larger number of followed cases has been accumulated. The total number of deaths due to metastases among the cases followed five years or longer is 239, or 48 per cent. This percentage increases to 66 per cent for the 200 cases followed ten

years or longer, apparently owing to the fact that many deaths from metastases occur between five and ten years after enucleation.

DISCUSSION

DR. WILLIAM M. JAMES, ST. LOUIS: The authors are to be complimented for presenting this material, which has been followed closely for nineteen years. Ten years ago 111 cases formed the basis of the present classification. Today 1,600 cases are presented. The classification has stood the test of time, and the added number of cases has verified the original conclusions.

Briefly, approximately 50 per cent of the patients have died in five years as a result of metastases; in ten years 66 per cent have died. The statistics presented show the great variation in the malignancy of the process. The incidence of the spindle cell type varies from 6 to 25 per cent. The epithelioid type of melanotic tumor is much more malignant.

There are only two points that I should like to bring out in discussing this paper. The first is that at the time the patient submits to enucleation the metastatic lesion has been planted in other areas of the body in 3 out of 5 cases. This fact is borne out by the ten year statistics. The second point that I should like to make is the value of this sort of report to the individual patient and to the physician. I think the variation in the individual reaction to the disease is so great that it is not advisable to give to the patient a gloomy prognosis. At least this should be withheld until the tissue has been examined histologically. That 66 per cent of the patients submit to enucleation after the lesion has spread indicates that present methods of diagnosis, the ability to impress patients concerning the fatality of the disease and present methods of treatment should be improved.

Tumors of the Limbus. COL. J. E. ASH, Medical Corps, United States Army, and HELENOR CAMPBELL WILDER, Washington, D. C. (by invitation).

Epithelial tumors are relatively common at the limbus. They are predominantly epidermal, and inclusions of epidermal elements are rather frequent. Occasionally other than epidermal elements are included, resulting in a mixed tumor or teratoma. In the material at the Army Medical Museum are 2 such tumors, 10 dermoids and 5 epithelial cysts. So far as our follow-up records go, there have been no recurrences or complications after excision. There are recorded in the registry isolated instances of lymphangioma, xerophthalmia, tyloma, keloid, xeroderma pigmentosa and lymphosarcoma. These and the instances of melanoma of the conjunctiva are not discussed.

Included in the study are 17 cases of epidermalization of the epithelium of the limbus with dyskeratosis. In some of these the changes were sufficient to lead to the suspicion of a tumor and excision and in some even to justify the dermatologists' expression "carcinoma in situ." There is no record of recurrence after simple excision.

Although in this group of cases actual neoplasm was not present, epidermalization of adjacent epithelium, with or without dyskeratosis, was seen in 12 of 28 cases of papilloma and in 35 of 48 cases of carcinoma, and it must be concluded that this is an important precursor to the

development of tumors. In 18 of the 28 cases of papilloma the tumor was excised, and there was only 1 recurrence. Eight eyes were enucleated because of extensive corneal involvement. In no case did papilloma recur persistently or metastasize.

Of the 48 cases of frank carcinoma, the tumor in 39 was squamous cell, in 8 papillary squamous cell and in 1 basal cell. Simple excision eradicated the tumor in 23 instances; in 2 instances additional excision was necessary, and in 23 cases enucleation was performed. Death was attributed to the tumor in only 1 of the 48 cases of carcinoma, and in this case the association was presumptive. It would seem that primary conservative surgical intervention followed by enucleation is justified if the recurrence is persistent. There seems to be no necessity for irradiation.

Treatment of Staphylococcic Blepharitis. Dr. Phillips Thygeson, New York.

The treatment of blepharitis remains unsatisfactory in spite of the many remedies proposed. By ordinary therapeutic methods it has been possible to produce improvement in most cases, but permanent cures have been rare. The present study concerns the treatment of blepharitis in which the predominating organism is hemolytic Staphylococcus aureus.

In the absence of experiments with the inoculation of human beings, the etiologic role of the staphylococcus in this, the most common type of blepharitis, can be assumed for the following reasons: 1. There is constant association of the organism with the disease. 2. The organism disappears when the patient is permanently cured. 3. The organism persists when the blepharitis recurs after treatment. 4. Specific anti-staphylococcic therapy has a favorable effect. 5. The organism occurs unilaterally in cases of unilateral blepharitis. Of the factors predisposing to localization and development of the organism, the most important in my series appeared to be seborrheic dermatitis of the face and scalp, acne vulgaris and acne rosacea. No evidence was obtained to indicate that vitamin A deficiency or refractive errors are significant factors.

In a study of local antiseptics applied in ointment or solution form, it was found that not one of the usual preparations, including the mercurial ointments, gentian violet and brilliant green, was effective in producing either bacteriologic or clinical cure. When local treatment was supplemented by the use of staphylococcus toxoid, approximately half of the patients became clinically healed, but in some of those whose cultures remained positive blepharitis recurred after periods varying from three to five months.

In certain cases of severe involvement chemotherapy consisting of oral administration of sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) and sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) was tried without permanent results, but sulfathiazole or sodium sulfathiazole ointment administered locally in from 1 to 5 per cent concentration produced rapid and dramatic results, which were far superior to those produced by any other preparations tried. While rapid improvement was obtained by using the drug once daily at night, the best results were obtained from application of the ointment intensively four to six times daily to both lid margins and the conjunctiva over a period of

several weeks. When used in conjunction with staphylococcus toxoid, sulfathiazole ointment gave a high percentage of clinical and bacteriologic cures.

DISCUSSION

DR. JAMES H. ALLEN, Iowa City: My experience in the treatment of staphylococcic blepharitis has been similar to that of Dr. Thygeson. Local therapy consisting of cleansing the lid margins and then applying the usual antiseptic ointments has resulted in temporary improvement. Similarly, irradiation applied locally to the lid margins has brought about temporary improvement. However, in all instances pathogenic staphylococci have persisted and exacerbations have occurred.

Staphylococcus Toxoid Treatments

Four Day Interval		
		First Strength
January	1	0.1 cc.
	5	0.2 cc.
	9	0.3 cc.
	13	0.4 cc.
	17	0.5 cc.
	21	0.6 cc.
	25	0.7 cc.
	29	0.8 cc.
		Second Strength
February	6	0.1 cc.
	10	0.2 cc.
	14	0.3 cc.
	18	0.4 cc.
	22	0.5 cc.
	26	0.6 cc.
March	2	0.7 cc.
	6	0.8 cc.
	10	0.9 cc.
	14	1.0 cc.
Seven Day Interval		
		Second Strength
March	21	1.0 cc.
	28	1.0 cc.
April	4	1.0 cc.
	11	1.0 cc.
	18	1.0 cc.

Noting the inflammatory changes in the conjunctiva which developed after the instillation of staphylococcus toxin, I began to treat staphylococcic blepharitis by immunization with toxoid. The lid margins were cleansed, and an antiseptic ointment was applied to them and to the conjunctiva in all cases for varying periods, but the patients continued to have symptoms, and the presence of pathogenic staphylococci persisted. The usual course of treatment is illustrated in the accompanying table.

In several cases the initial injection of toxoid was followed in twenty-four or forty-eight hours by an exacerbation of the blepharitis. However, the symptoms subsided after the second or third injection and in turn were followed by rapid improvement. In the remaining cases gradual improvement began within a few days after the initial injection of toxoid. In practically all cases the symptoms diminished to such an extent that the patients considered themselves cured after approximately

ten injections. As a consequence, several patients failed to return for the complete course of immunization until after a few weeks, when they suffered exacerbations of the blepharitis.

In some cases there was improvement in the condition after the injection of toxoid, but the symptoms were not entirely relieved. Slight crusting and redness of the lid margins persisted, and some conjunctival irritation remained. In another small group of cases symptoms recurred several weeks after the course of immunization injections had been completed. In both groups pathogenic staphylococci were cultured from the secretions of the conjunctiva or of the lid margins.

As these therapeutic failures became apparent, I discovered an explanation which might account for part of them. I had kept track of the staphylococcus antitoxin titer of the blood of the patients. Before treatment the titer was less than 1 unit per cubic centimeter of serum. In the majority of cases, one week after the last injection this titer had risen to 4 or more units per cubic centimeter of serum. The highest titer was 17 units per cubic centimeter of serum. But in the cases in which symptoms had persisted or in which exacerbations had occurred, the injections of toxoid had failed to produce a titer of more than 3 units per cubic centimeter of serum. Further injections of toxoid did not increase the titer in these cases.

Therefore, supplementary therapy was sought. Sulfanilamide was found to be ineffective by either oral or local administration. Sulfathiazole administered orally in doses of 0.5 to 1 Gm. three or four times a day for ten days resulted in temporary improvement, but sooner or later exacerbations occurred. However, it was found that the local application of a 5 per cent ointment of sulfathiazole three to six times a day resulted in rapid improvement. During the past year approximately 40 patients have been treated for one month by this method alone. In 2 cases the organisms persisted and an acute exacerbation of symptoms occurred during treatment. In 2 cases exacerbations occurred several weeks after the treatment was discontinued. In 2 cases contact dermatitis and keratitis developed owing to sensitivity to some constituent in the ointment. In the remaining cases the symptoms were negligible within seven to fourteen days after the institution of sulfathiazole therapy, and pathogenic staphylococci were not demonstrable in cultures after fourteen days.

At the present time I am observing a group of patients who are being treated with both toxoid and a 5 per cent ointment of sulfathiazole, and although it is impossible to draw definite conclusions, it seems likely that this combination is the treatment of choice.

In conclusion, there is one point about the treatment of staphylococcic blepharitis which I believe should be emphasized, i. e. the local treatment of the meibomian glands and infections of the follicles of the lashes. Secretion from the glands should be expressed until normal secretory function has been reestablished. Otherwise the glands may harbor pathogenic staphylococci and exacerbations of the blepharoconjunctivitis result.

DR. FREDERICK C. CORDES, San Francisco: During the time that Dr. Thygeson was doing some of the work reported in his paper, Dr. Hogan, of the University of California Medical School, had the privilege

of working with him in his laboratory and wrote to his associates about it. Since that time we have employed sulfathiazole in cases of staphylococcic blepharitis.

It is surprising to note the large number of cases of persistent chronic blepharoconjunctivitis in which hemolytic Staphylococcus aureus has been recovered on culture. The response to the local application of sulfathiazole ointment has been equally surprising.

The treatment consists of massaging a 5 per cent ointment of sulfathiazole with an oxycholesterol-petrolatum base into the conjunctival sac. The patient is requested to return the following day, and if there has been no reaction the drug is prescribed to be used one to four times a day. The patient is instructed to place a small amount of the ointment into the conjunctival sac and massage it in thoroughly. He is also advised to watch for swelling, increased redness and itching as signs of sensitivity to the drug. If these occur the use of sulfathiazole ointment is discontinued, and one of the other sulfanilamide derivatives is tried. In our experience allergic reactions are fairly frequent.

Recently a base consisting of 75 per cent petrolatum and 25 per cent hydrous wool fat has been used, as Dr. Thygeson has suggested. Failure to pulverize the powder sufficiently results in considerable irritation when the drug is used. It is well to estimate the smoothness of the ointment by placing some of it on the back of the hand or between the fingers before giving it to the patient to use. It is also advisable to be certain that the pharmacist to whom the patient takes his prescription understands thoroughly how to compound the ointment. Even though the vision is blurred for a short time after the ointment has been applied, the unguent base seems to be more effective than the water-soluble bases.

It is also desirable to continue to apply the drug for some time after the clinical symptoms have disappeared and until cultures negative for pathogenic staphylococci are obtained.

For patients who are not sensitive to the drug it seems best to apply it more frequently in order to obtain a more constant local concentration.

This therapy has also been beneficial in the treatment of small marginal corneal ulcers which develop in the course of mild staphylococcic blepharoconjunctivitis. In addition, it has materially reduced the recurrence of such ulcers when it has been continued over a long period of time.

The following case is rather typical:

Mrs. P., aged 24, had been under treatment intermittently for six years because of persistent blepharitis that did not respond well to any of the therapeutic measures used. Recurrences were numerous. Conjunctival scrapings and cultures revealed hemolytic Staphylococcus aureus. Sulfathiazole ointment was prescribed, and after three weeks the condition was clinically cured. The patient continues to apply the ointment at night, and to date there have been no recurrences.

As for toxoid, I have found it to be helpful in several cases but have not had sufficient experience with it to draw any conclusions. A logical procedure seems to be the local application of sulfathiazole together with injections of toxoid in order to stimulate the general response.

I believe that Dr. Thygeson's method of treatment will prove to be a valuable aid in the treatment of persistent staphylococcic blepharitis.

DR. PHILLIPS THYGESON, New York: I wish to thank Dr. Allen and Dr. Cordes for their discussions.

Irritation from the ointment I think is a significant factor. If sulfathiazole is used, the crystals themselves are definitely irritating unless they are ground up well and sifted through fine mesh so that irritating particles are removed. Sodium salt, which is in powder form, can be used and seems to be equally effective. It is slightly alkaline but not sufficiently so to cause irritation. The salt is probably less stable than the acid, but it seems to last during the life of the ointment.

As for the relative value of the use of toxoid in conjunction with local therapy, I certainly feel that the local therapy is of first and the toxoid of secondary importance. I recently had an opportunity to observe what amounted to epidemic blepharoconjunctivitis in patients confined in a mental institution near New York. Rather surprisingly it was a mixed type of infection, diplobacilli and Staphylococcus aureus being cultured. All previous medication, including mercury and zinc salts, had failed utterly to check the condition. Sulfathiazole ointment proved effective in eliminating symptoms although not particularly effective in preventing recurrences. If it had been possible to use toxoid, I think the incidence of recurrence would have been much less.

Evaluation of Operations for Glaucoma. DR. BRITTAIN F. PAYNE, New York

Ideal postoperative results for the relief of glaucoma are so rarely seen in the laboratory that histologic studies of the material at the New York Eye and Ear Infirmary were made to determine the causes of failure.

Examination of most of the globes showed that the operative wounds were anterior to the angle of the iris and contained remnants often of the iris and occasionally of the ciliary processes. Epithelial downgrowths were particularly evident after cataract extractions, and in many instances some of the lens capsule was included in the wound.

Cases of glaucoma complicated by diabetes were characterized by migration of pigment into the pectinate spaces, by fibrosis and by synechias. When iridocyclitis involved the eye, the microscopic picture was somewhat altered, often showing more organized exudate and greater synechias.

The type of operation performed did not change the microscopic picture appreciably. Simple paracentesis often showed tags of degenerated uvea, pigment, organized exudates and hemorrhage. Iridectomy seldom included the root of the iris, and the synechia was unchanged by the operation. Fistulous tracts were found in some cases in which an anterior sclerectomy or a trephining was performed. Delayed infection was noted in a few cases in which trephining was the treatment. Iris inclusion operations showed fibrous replacements with little evidence of filtration.

It was concluded that operative failures were due to poor filtration and inflammatory reaction in most cases.

DISCUSSION

DR. PETER KRONFELD, Chicago: Being a stickler for scientific terms, I should first like to point out that the title which Dr. Payne has chosen for his paper does not do justice to the work on which the paper is based. Evaluation of operations for glaucoma from the standpoint of the clinician is something that ophthalmologists all do to a greater or lesser extent. Dr. Payne has studied the effect of antiglaucomatous operations from a different point of view and on unusual material. He has endeavored to determine what factors are entailed in antiglaucomatous operations that may lead to enucleation.

Probably for good reasons Dr. Payne has omitted from his paper all information pertaining to the history and the clinical findings in his cases. It may, however, be presumed that the enucleations were done because of the presence of an intolerable state of decompression that had not been relieved or even had been aggravated or brought on by an antiglaucomatous operation. Thus the operations which had been performed on the eyes of Dr. Payne's patients not only failed to reduce the intraocular tension but failed to bring about a state of fair ocular comfort. The pathologic examination of these eyes may, therefore, be expected to show why they remained painful and irritated, in addition to showing why no new channels of outflow for intraocular fluid were established.

With regard to the individual operations which Dr. Payne has mentioned: Although I have advocated paracentesis as a diagnostic procedure for certain forms of glaucoma and as a therapeutic procedure for others, I have advised against its therapeutic use for acute uncompensated and for chronic compensated glaucoma because the severe hypertensive reaction which is set up by paracentesis tends to aggravate the existing condition. I suppose that the two eyes mentioned by Dr. Payne (the two eyes for which enucleation became necessary after paracentesis) belonged in the group of eyes affected by these conditions.

In describing his case of glaucoma in which obstruction of the central vein was associated, Dr. Payne commented that the eye was removed because the pressure was not relieved by the operation and because the iridectomy failed to include the base of the iris. Gonioscopic observations in such cases have shown that the tension may be elevated before a peripheral anterior synechia is formed. Removal of some of that synechia would, therefore, not guarantee a return of the tension to normal. In other words, I rather suspect that in this instance Dr. Payne was dealing with a case in which the peripheral anterior synechia was probably not the cause but the result of glaucoma.

In discussing iridencleisis, Dr. Payne dealt with one of the most typical reasons for removal of an eye after an antiglaucomatous operation, namely inclusion of ciliary processes and a portion of the lens in the operative wound. I believe that the mechanism of this inclusion in most instances results in too rapid a decompression at the time of the operation. Such a result, of course, happens more easily if the intraocular pressure is high at the time of the operation. If the surgeon exerts pressure on the eye with the fixation forceps and with the instrument that he uses to cut through the sclera or the cornea, and especially if the decom-

pression takes place rapidly, a prolapse of the ciliary processes and of the lens into the wound is likely to take place.

In summary, while the advent of the gonioscope has made the clinical evaluation of operations for glaucoma infinitely more accurate, there still is a great deal to be learned from pathologic studies of the type that have been described.

Problem of Preventing Partial or Total Loss of Vision in Patients with Glaucoma Treated at Ophthalmic Clinics: Suggestions for Remedial Measures. DR. MARK J. SCHOENBERG, New York.

It is generally admitted that every patient with glaucoma is a potential victim of blindness and that in a certain percentage this could be prevented by early diagnosis and proper treatment.

Although the earlier diagnosis and control of glaucoma have been made possible by the discovery of a number of methods and procedures, such as ophthalmoscopy, quantitative perimetry, angioscotometry, use of tonometric graphs, provocative tests, slit lamp microscopy activities of social workers and educational campaigns, the utilization of these discoveries has lagged behind in the very quarters to which most of the patients with glaucoma are likely to apply for advice and treatment.

It seems that in ophthalmology, unlike many other applied sciences, the distance in time between a discovery and its universal application —especially in ophthalmic clinics—is in some instances as much as half a century.

This situation, which has a direct bearing on the results of efforts to prevent partial or total loss of vision from glaucoma, is the reason for submitting this contribution.

The paper consists of two parts:

Part 1 reports on surveys of records on glaucoma in three large New York ophthalmic clinics. The findings are summarized, and the weaknesses of the present system of diagnosis and treatment of glaucoma prevailing in clinics are pointed out.

Part 2 gives an outline of suggestions for remedial measures. The attempt is made to bring about (*a*) the elimination of objectionable features and (*b*) the adoption of definite measures for more efficient methods of handling the problems of patients with glaucoma.

With these thoughts and this program in mind, the National Society for the Prevention of Blindness has undertaken a campaign for the reduction of the percentage of partial or total loss of vision from glaucoma and invites the cooperation of ophthalmologic organizations to carry out this task.

DISCUSSION

DR. THOMAS D. ALLEN, Chicago: This has been a most interesting and instructive paper. It assures a continuation of the forward looking policies adopted by the American Society for the Prevention of Blindness Dr. Schoenberg has gone over the situation so thoroughly that there is nothing more to be said except to reiterate and emphasize some of his points and possibly add a few words about the steps now being taken in Chicago to get remedial measures started.

Some years ago Dr. Gradle and one of his assistants reviewed the situation with respect to glaucoma at the Illinois Eye and Ear Infirmary, where there are probably seen more new patients per day than in any similar medical institution in Chicago. They found that only 4 per cent of the patients with glaucoma were being properly followed and that 96 per cent were lost track of.

After consultation with the six separate eye services in Chicago it was agreed to have a separate glaucoma clinic established in order not to lose track of these patients. It was obvious that neither the six attending ophthalmologists nor their associates nor their adjuncts could give the time to explain adequately to each patient the condition present in his eyes and the necessity of frequent examinations; nor could they go to the patient's home and study the social situation or the psychologic situation, which often play a tremendous role in this disorder.

The glaucoma clinic was established three years ago, and now the situation is completely reversed. Ninety-six per cent of all patients suffering from glaucoma who come to the infirmary are adequately followed and can be accounted for. Only 4 per cent are lost track of. This is a tremendous improvement. It means that the incidence of blindness from glaucoma in the area served by the clinic is probably being substantially reduced. The work is accomplished by the cooperation with the glaucoma clinic of the infirmary's six separate services.

As soon as a diagnosis of glaucoma is made or glaucoma is suspected, the patient is referred to the glaucoma clinic, where a fairly complete work-up from the ophthalmologic standpoint is made of the case, including the taking of a history, general physical examination (this is not adequately done as yet, but progress is being made), visual tests, refraction, repeated tonometric examinations, gonioscopic examination and the taking of fields. A carefully selected, trained social worker visits the patient's home and inquires about the social, financial and psychologic situation. She is in close touch with other social agencies in the city, frequently calling on them for assistance. The social worker explains to the patient the reason for careful and repeated examinations. She explains the reasons for medication and shows the patient how the instructions of the ophthalmologist can be carried out in the home. She gives the patient a definite appointment for consultation, and if he does not appear she sends a card, telephones or visits his home. If the patient has gone to a private physician, her duty is ended. If the patient has not carfare to go to the clinic, she supplies that. She sees that the patient has medicine and that he knows how the drops are to be used. She assists in having him referred to a general clinic or to a psychiatric clinic if this is necessary. She helps to solve other problems in the home.

The next step, as I understand it in reading Dr. Schoenberg's paper, is for all ophthalmologists in clinics in Chicago to organize and form a central clearing house. Each of the clinics is to have a similar glaucoma service, so that should a patient, for example, leave the clinic of the Illinois Eye and Ear Infirmary and go to the Northwestern University ophthalmic clinic or to the University of Chicago ophthalmic clinic he will be adequately followed. This can be done only if the ophthalmologists rise above petty jealousies. It can be done only if they take great interest in the problem and in each patient who is a part of the problem. It can be done better if there is established a uniform system of keeping

records which contain all the necessary data so that the roving patient will not have to endure ten or fifteen hours of unnecessary preliminary work-up each time he chooses to change from one clinic to another. It can be done best if there is not too frequent change in the medical personnel or in the personnel of social service workers.

One of the great values of the glaucoma clinic at the Illinois Eye and Ear Infirmary has been the fact that the residents and the interns have become "glaucoma conscious." They are able to investigate a patient thoroughly for the earliest signs of glaucoma, for the so-called "pre-glaucomatous state."

Again let me congratulate Dr. Schoenberg on his magnificent presentation of a subject which is so vital to the ophthalmologist as a physician and as a citizen. I hope that the matter will be given serious consideration by ophthalmologists throughout the country to the end that there will be fewer citizens condemned to blindness by sins of omission on their part.

DR. C. GREGORY BARER, New York (by invitation): I wish to congratulate Dr. Schoenberg on his presentation of a subject that everybody realizes is a major problem.

In New York city, under the direction of the National Society for the Prevention of Blindness a glaucoma committee on a demonstration basis has been organized. It is composed of a representative of each ophthalmic clinic in metropolitan New York. These various ophthalmologists are cooperating in forming a local glaucoma unit to supervise the glaucoma work in their own institutions. The glaucoma committee does not dictate what the management of each individual case will be. That is left to the discretion of the attending ophthalmologist. The committee has furnished a uniform record form to contain a minimum amount of data, so that at least part of the work done will be standardized.

The plan is not the ideal one by any means, but under the circumstances, since it is impossible to organize and have a functioning glaucoma clinic in each institution, it is the next best. It is felt that with it in operation better work on glaucoma will be done and the hoped-for result obtained.

MISS RUTH EMERSON, Director of Social Service Department, University of Chicago Clinics, Chicago (by invitation): In 1937, Dorothea Chickering, in fulfilment of the thesis requirement in the School of Social Service Administration, made a survey of the patients with glaucoma registered over a ten year period in the ophthalmic department of the University of Chicago Clinics. Prof. E. V. L. Brown, chief of service, and the Social Service Department were eager to know how adequately the clinic was meeting the needs of its patients. In 1940 Frances Ross, social worker in the ophthalmic service, brought this study up to date. The findings and the recommendations which emerged from these studies are for the most part similar to those which Dr. Schoenberg reports. I should like to comment briefly on the social component which is inherent, although perhaps not obvious, in certain of his recommendations.

The University of Chicago studies showed that one of the most important and frequent causes of a patient's failure to continue under regular treatment was his feeling that the clinic was not interested in him —that the atmosphere and the staff were too impersonal. Point five

refers to the training of the younger men in the art and science of diagnosis and treatment of patients suffering from glaucoma. Implicit in the art of medicine is the recognition of human personality. This requires both understanding the individual and skill in knowing how to motivate him to play his role in medical care and life so that he may live with some degree of psychologic comfort. The medium used by the physician to create the effect he wishes, namely the active participation of his patient in carrying out measures to safeguard his eyesight, must be applied differently with each patient. Until there is consideration of the patient as a person throughout his clinic experience it is probable that many patients will not complete their treatment.

In recommendations 5, 7 and 9, attention is called to the importance of recording the data necessary for proper treatment, of having these available to the surgeon prior to operation and of having conferences regarding patients seriously threatened with blindness. The execution of these recommendations in respect both to written reports and to the discussions requires consideration of the individual patient, the meaning which his illness has to him, his social setting and his relations to family, social group and work. The medical social worker contributes to the clinical picture her findings and experience regarding the case; this leads to a more refined evaluation of factors in respect to treatment.

Dr. Schoenberg did not present his basic assumptions in regard to clinic practice as a background against which to consider his recommendations, but I do not think it irrelevant to consider the philosophy of clinic care.

Boards of trustees of hospitals, public and private, have brought together personnel and equipment which physicians affirm are essential in giving patients the full benefit of modern medicine. The boards entrust to the clinicians the use of these resources in the hope that, so far as possible, patients may regain health and be better able to function effectively in everyday life. I heard a physician put it this way to his students: "Remember, the only persons who have rights in the hospital are the patients. The rest of us have privileges and related obligations." Research and education are not disassociated from the best care of the patient.

The medical institution is to serve persons who are ill. It is necessary, therefore, to understand the person who is to be served. The patient looks to his physician with confidence, and there can be therapeutic values in this relationship. But all who work in the medical institution may be corrupted at times by its highly organized aspects, and it is not in criticism of the necessary procedures but because of human susceptibility to routine that I would call attention to the importance of perspective. Perspective is essential if the human values inherent in medical care are to be developed appropriately, values essential to the art of medicine.

One is inclined to think that it would take too much more time to give the patient an opportunity to tell something about himself and his concern about his ocular condition, and therefore, doctors, nurses and social workers often short-circuit their interviews with the patient by dogmatically telling him what to do and how to do it, forgetting that often the patient does not understand, does not sense why it would be to his

advantage to do what is suggested, or does not know how to go about carrying out the recommendations. I am reminded of the patient with glaucoma who said a little hopelessly, "The doctor said I should not worry and I worry because I worry." It is probable that in the long run time will be saved, and a considerable amount of time, if the patient in his early association with the clinic is given an opportunity to reveal something of himself as an individual who suffers from symptoms rather than a mere spokesman of symptoms. It has been clearly demonstrated in clinics for venereal disease that the patient who talks with his doctor and social worker about himself, his condition and his treatment at the time of his first or second visit has increased confidence in the clinic, is cooperative and comes more regularly for treatment.

A philosophy of service requires of all who are working together in a hospital emotional maturity so that they may be tolerant, and accepting of the patient, and able to feel with him, not like him. As Dr. Allen has pointed out, there is no place for jealousy. The medical social worker came into the hospital because clinicians wished to have fuller data regarding the patient as a social being and to help the patient work through problems within his personality or environment which thwart his deriving full benefit from the doctor's skill and the institution's resources. As a professional colleague, she can extend the doctor's understanding of his patient and the patient's understanding of his condition and of the doctor. But at the core of medical care there must be mutual acceptance and confidence between physician and patient.

In other words, I believe that the application of Dr. Schoenberg's recommendations is dependent on a basic philosophy which recognizes the integrity of the patient and asks that all who work in the clinic adjust to the patient rather than expect or ask him to adjust to the clinic. The social worker is eager to make her contribution, but she, like other members of the medical team and like the patient, looks to the physician to give leadership and to set the standards of individualized services to patients.

MRS. ELEANOR BROWN MERRILL, Executive Director, National Society for the Prevention of Blindness, New York (by invitation): I am glad to have this opportunity to discuss Dr. Schoenberg's paper from the standpoint of the National Society for the Prevention of Blindness, whose function is to study the extent of blindness due to glaucoma and to keep cognizant of opportunities of preventing loss of vision.

More accurate accounting of data in recent years changes the previous estimate of 130,000 blind persons in the United States to 200,000; of these, the number whose blindness is due to glaucoma is believed to be approximately 20,000. This estimate is based on data on the causes of blindness among adults receiving pensions for the blind in eighteen states, which indicate that 11 per cent are blind from glaucoma.

The incomplete reporting of five or ten years ago does not allow for comparative statements regarding the incidence of blindness from glaucoma today. There is no evidence of decrease; there is some reason to believe that the incidence of this causal factor is on the upgrade. Yet Dr. Schoenberg calls attention to the methods and procedures through which control of glaucoma is made possible. He refers to the lag in universal

application which has to a great extent prevented successful accomplishment. It is interesting in this connection to recall the lapse in time between the discovery and the application of prophylaxis in relation to ophthalmia neonatorum. The marked decline in incidence of blindness from ophthalmia neonatorum over the past thirty-five years encourages one to think that through concerted action and ophthalmologic leadership a much lower percentage in blindness from glaucoma may be reached.

Dr. Schoenberg states that every patient with glaucoma is a potential victim of partial or total loss of vision and that in many cases such loss is preventable through early diagnosis, persistent treatment and the patient's understanding cooperation. He enumerates some of the weaknesses which lie back of failure to retain useful vision. As representing an educational organization, I note particularly the point referring to lack of understanding among patients, without which satisfactory treatment or control is impossible. All workers, both lay and professional, feel a responsibility in this connection. The importance of the physician-patient relation, the need of social case handling and the wisdom of carefully planned instruction are all evident. In one hospital recently visited by the society, classes have been organized for patients with glaucoma, who respond intelligently to lectures conducted by members of the staff.

I wish to emphasize what Dr. Schoenberg mentions later in his discussion—education of the public. Not only must the public be made to see the need of supporting both morally and financially the services required for successful handling of patients with glaucoma, but it must be made aware—without a terrorizing campaign—of what may be early indications calling for careful medical examination. To this end, the National Society for the Prevention of Blindness, with guidance and cooperation from ophthalmologists, can take a leading part.

Let me reiterate Dr. Schoenberg's wish for a forging ahead in a joint universal effort to prevent blindness from glaucoma.

Dr. Harry S. Gradle, Chicago: I believe that the measures to be employed in preventing total loss of vision in patients with glaucoma extend further than the mere establishment of clinics and central registeries for these patients. This belief has been held in Chicago for some time and it has been felt that the establishment of clinics for the treatment of glaucoma is merely the first step.

A patient who comes to a glaucoma clinic is going to be well cared for, and every effort will be made to prevent blindness from this disease. But how is the patient to be contacted? He does not know what glaucoma is, and he cannot diagnose it himself. Many patients who have an ocular complaint are suspicious of the presence of glaucoma. The majority of those patients who are suspicious do not have glaucoma at all. Who is going to see the patient first and inform him of the possibility of the presence of glaucoma or direct him to an ophthalmologist who is capable of administering the proper treatment during the early stages of the disease? The treatment cannot be administered by a lay person. An optometrist cannot be expected to diagnose glaucoma and then send the patient to an ophthalmologist. The public health nurse does not come in contact with persons in the first stages of glaucoma. The general practitioner is the first person with any diagnostic skill to see the patient.

The general practitioner who cannot diagnose glaucoma is a menace to the future of a patient suffering from this disease, and it is the duty of ophthalmologists to inform general practitioners regarding the early features of glaucoma and regarding the possibility of its existence in any patient past the age of 30. The general practitioner should include in the routine of his general physical examination of each patient a sufficient examination of the eyes to elicit at least the manifest symptoms of glaucoma in its early stage. This procedure could be facilitated if ophthalmologists would contact general practitioners and explain to them the early manifestations of glaucoma. The general practitioners are not going to come to the ophthalmologists. In Illinois a plan is being developed which it is hoped will be put into effect during the course of the next few months. The work is financed by the foundation which financed the glaucoma clinic. Groups of ophthalmologists will visit all the county medical societies in Illinois, the groups being composed of the ophthalmologists of each county plus ophthalmologists of Chicago. They will demonstrate the aspects of glaucoma to the general practitioners and teach them how to diagnose the disease in its early stage.

There is every reason to believe that such campaigns will help in teaching general practitioners how to diagnose glaucoma in its early stage, with the result that many patients may be spared impending blindness from glaucoma that has been unrecognized over too long a period of time.

DR. MARK J. SCHOENBERG, New York: In the first place I should like to express my enthusiasm for the wonderful work that is being accomplished in the glaucoma service at the Illinois Eye and Ear Infirmary. I had the opportunity of observing it, being permitted by Dr. Gradle to visit the clinic under the able guidance of Dr. McGarry.

Also, I have enrolled in the course on glaucoma, and I am happy to say that it is excellent. The exciting thing about what I have seen happening in some glaucoma clinics in Chicago is that it corroborates what I have been thinking and talking about for some time, namely the change in the attitude which must be adopted in dealing with patients who have glaucoma. Ophthalmologists are no longer satisfied with the policy of *laissez faire* but want to do something about this problem, and this something is happening in Chicago. I am confident that Dr. Gradle, with his great executive ability, will organize his own clinic and also all the clinics in the city of Chicago.

The same thing is happening in greater New York. A group of men representing eighteen ophthalmic institutions in New York city has been organized under the name of a committee on glaucoma.

I appreciate Miss Emerson's kindness in discussing this paper, and I want to assure her that interest in the patient himself as well as in disease has been also one of my concerns, and it certainly will not be forgotten.

Centuries before the time of Christ, Socrates spoke about the importance of treating the human being as a whole, yet only during the past few decades has this idea been taken up seriously.

I am glad that Dr. Gradle has brought up the question of the role of the general practitioner in the campaign of preventing blindness from glaucoma. Last winter I had the pleasure of discussing this subject with Dr. Gradle, and we both agreed that there is a great deal to it. In

New York state a step has already been made in the direction of cooperation between the general practitioner and the ophthalmologist, and I hope that within a few years projects pointing in this direction will be carried out all over the country. A paper on this subject was presented at the annual meeting of the New York State Medical Society and will appear shortly in the *New York State Journal of Medicine*.

Bacillus Pyocyaneus Ulcer: Report of Three Cases; Results of Sulfapyridine Therapy in One Case. Dr. C. W. Lepard, Detroit.

This report includes the clinical experience in 3 cases. In 2 cases, in spite of all treatment beginning panophthalmitis developed, and enucleation of the eye was carried out. The third patient, who was treated with sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) responded promptly and recovered. This final vision was 20/20 with glasses.

Sulfanilamide was used late in the course of the disease in the second case. While the course was distinctly modified, the infection progressed, and finally the globe had to be removed.

The use of sulfapyridine in the third case is described in detail. The importance of early identification of Bacillus pyocyaneus in a direct smear of material from the ulcer and immediate use of the drug are emphasized.

The organism was recovered from a solution of fluorescein used prior to the time of infection in the third case.

DISCUSSION

Dr. S. Hanford McKee, Montreal, Canada: Ulcers of the cornea due to B. pyocyaneus infection have been reported from time to time. This micro-organism has also been isolated in cases of atypical keratitis, dacryocystitis, metastatic inflammation of the orbit, ophthalmia neonatorum and other conditions. It is perhaps the most virulent micro-organism which attacks the cornea, and usually when it does so it leads to complete loss of the eye. It produces powerful toxins which can damage the epithelium and thus allow the bacillus to enter the cornea proper. Rapid sloughing of the cornea usually occurs, followed by panophthalmitis. While B. pyocyaneus is considered a saprophyte, it must be remembered that different strains of the micro-organism vary considerably in virulence. In a number of cases in which the virulence of B. pyocyaneus was tested, it was found to be high.

Dr. Lepard's presentation is timely in (1) reporting the fact that the ever present bottle of fluorescein may carry a virulent micro-organism and in (2) reporting the successful treatment of a patient with sulfapyridine. In this connection, I should like to report 3 cases in which the ulcerated cornea was treated with chemotherapy.

Case 1.—S. T. J., a laborer aged 53, was suffering from a severe serpiginous ulcer of the right eye which developed after trauma. Different local applications did not affect the progress of the infection to any extent. Sulfanilamide was administered orally. Twenty-two and a half grains (1.45 Gm.) was given every four hours until 645 grains (42 Gm.) had been given over a period of five days. The ulcer healed, but a large scar remained. Optic iridectomy would have made the eye useful, but the patient would not submit to this procedure.

CASE 2.—T. C., a small boy aged 7 years, was brought to me in April last with a severe inflammation in the left eye. He had been struck in the eye some days previously while playing with older boys. Smears and a culture of material from the eye revealed the gonococcus. A large central lesion of the cornea (6 by 6 mm.) was present, which stained deeply with fluorescein. Seven and a half grains of sulfanilamide (0.51 Gm.) was prescribed to be taken orally, and sulfanilamide powder was dusted into the conjunctival sac. The patient was then given 15 grains (0.97 Gm.) of sulfanilamide a day for three days. Use of the drug was discontinued because there was no further discharge from the eye. The patient made a complete recovery.

CASE 3.—Mrs. E. J., aged 48, had had her right eye removed, because of an ulceration of the cornea, a few days previous to the time that I saw her. My examination revealed a severe ulcer, of the Mooren type, in the left eye. The ulcerated surface extended peripherally almost entirely around the cornea, and only a small area free from infection remained in the region below the cornea. Hypopyon was not present. The patient complained of severe pain, some of which no doubt was due to iritis. Fifteen grains of sulfanilamide four times daily was administered orally. Salicylates were also given daily, because the patient suffered from rheumatoid disease. Three per cent sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) ointment was applied locally to the eye night and morning, and a bandage was applied over the eye. In five days the patient had been given 300 grains (19.5 Gm.) of sulfanilamide, and her condition was so satisfactory that use of the drug was discontinued. The ocular condition was completely healed a few days later, at which time she returned to her home. There was, of course, a definite scar of the periphery of the cornea, but otherwise the eye was completely well.

DR. HAROLD H. JOY, Syracuse, N. Y. (discussion read by DR. D. F. GILLETTE, Syracuse, N. Y.): The malignant course of B. pyocyaneus infection of the cornea is probably due to the rapid migration of the organisms from the site of infection to other parts of the cornea. Local therapy usually is ineffectual because by the time treatment is instituted the organisms have already invaded the normal tissues and are inaccessible.

The striking result obtained with the use of sulfapyridine in Dr. Lepard's third case indicates that chemotherapy may be the solution in combating this eye-destroying infection. However, one must not forget that there is a great variation in the virulence of different strains of B. pyocyaneus.

Theoretically, sulfanilamide and its derivatives fulfil the requirements for treatment of this type of infection. After oral administration, both sulfapyridine and sulfanilamide rapidly penetrate into the corneal stroma and do not have a deleterious effect on normal tissue and fluid elements. Furthermore, the drugs are antibacterial against certain organisms in concentrations which can easily be attained by therapeutic administration.

In view of these facts, it is necessary to determine whether sulfapyridine is antibacterial against B. pyocyaneus and whether in a large number of cases its administration will favorably affect the course of infection produced by a virulent strain of the organism. Owing to the infrequent occurrence of this type of infection one must depend on experimental inoculation of animals to determine the efficacy of therapy.

I wish briefly to report experimental results which will be published in full detail in the ARCHIVES OF OPHTHALMOLOGY.

(Dr. Joy's article, "Treatment of Experimental Bacillus Pyocyaneus Ulcer of Cornea with Sulfapyridine," appeared in the June issue of the ARCHIVES, page 1135).

DR. CARL J. RUDOLPH, South Bend, Ind.: One week ago last Monday morning I eviscerated two eyes that had been infected by B. pyocyaneus. Following keratitis and severe ulceration of the cornea, panophthalmitis developed on about the twelfth day in the older patient, a man 52 years of age, and on about the fourteenth day in the younger patient, a man 30 years of age.

In reviewing the recent literature concerning sulfanilamide and its derivatives, I could find no information regarding their curative or effective properties in the treatment of infections due to B. pyocyaneus. Regardless of this and although I realized how hopeless topical application is in the treatment of infections due to B. pyocyaneus, I supplemented this therapy in the case of the younger patient with the oral administration of sulfathiazole. Seventy-five grains of sulfathiazole (4.87 Gm.) was given the first twenty-four hours, and the medication was retained. The sulfathiazole which was given after the first twenty-four hours was not retained, because the patient vomited; so I discontinued oral therapy. I continued to apply 5 per cent sulfathiazole ointment locally over a period of four days, but it had no effect whatever.

Two days after the first (or younger) patient had consulted me, the second (or older) patient came into my office. I immediately referred him to the medical laboratory for a smear and a culture, which I had done in the first case on the second visit. The culture in each case was positive for B. pyocyaneus. I used a different method in the second case. The patient was older, and his resistance to the infecting organism evidently was not so great.

After consulting a pathologist and learning that elsewhere in the body B. pyocyaneus is amenable to treatment with acetic acid and alcohol, I applied glacial acetic acid on the end of a toothpick applicator to the ulcerated area and neutralized this with a 70 per cent solution of alcohol. On about the tenth day, I performed a delimiting keratotomy on the younger patient where the ulcer remained confined to the lower two thirds of the cornea. The ulceration had spread over the entire cornea of the older patient, and on the eighth day I did a paracentesis. This in no way checked the condition. So, after thirteen days' treatment of the older patient and fifteen days' treatment of the younger patient, I found it necessary to perform an evisceration. Late in the course of the treatment in each case 4 Gm. of sulamyd (acetylsulfanilamide) was administered orally, but it did not prove effective.

I did not choose to perform an enucleation for fear of opening up the vaginal space which communicates with the arachnoid space of the brain. The eyes of both patients are healing satisfactorily. I regret that I had not heard of the chemotherapeutic value of sulfapyridine. I do not believe that any articles concerning it appear in recent literature, and I do not believe that commercial drug companies claim that they have produced a sulfanilamide derivative that has any effect on B. pyocyaneus.

DR. EDMUND L. COOPER, Detroit: Dr. Lepard is to be complimented on so successfully treating an ulcer of the cornea due to B. pyocyaneus. It is rare that an eye infected with this virulent organism is saved, and to be able to bring such a case to so satisfactory a conclusion is indeed an achievement.

Some months ago Dr. Howell L. Begle, of Detroit, and I had the opportunity of seeing 4 patients with corneal ulcer due to B. pyocyaneus. They were men industrial workers, each of whom had been treated for a foreign body in the cornea. The offending organism was recovered from each ulcer and from a bottle of a 1 per cent solution of fluorescein from which each had been treated.

CASE 1.—The patient when first seen had a pinhead-sized ulcer at the site of the foreign body. This was treated in the customary manner with instillations of 1 per cent atropine sulfate solution, instillations of 1:1,000 aqueous solution of merthiolate, irrigations with boric acid solution and hot applications; but in spite of treatment the ulcer spread rapidly. Within twelve hours it had involved the whole cornea and a large hypopyon had developed. At this time the bacteriologic diagnosis of ulcer due to B. pyocyaneus was suspected, and an initial dose of 45 grains (2.92 Gm.) of sulfathiazole was given, followed by 15 grains every four hours around the clock. This dosage was calculated on the basis of ⅓ grain (0.02 Gm.) per day per pound (0.5 Kg.) of body weight. (The same basis was used in calculating the dosage for the other 3 patients. In all 4 patients the level of the drug in the blood was maintained between 6 and 11 mg. per hundred cubic centimeters.) After the second day, sulfapyridine was substituted for sulfathiazole. Progress of the ulcer stopped soon after the administration of the initial dose of sulfathiazole, and involvement of the eyeball was limited to the anterior segment. However, severe secondary glaucoma developed, light perception was lost and the eye was subsequently enucleated.

CASE 2.—The patient was seen two days after the removal of a foreign body from his eye. There was an ulcer involving one third of the cornea. The edges were undermined, and the base was covered with tenacious discharge. Chemotherapy with sulfathiazole was started in addition to the customary local treatment. After the first day, sulfapyridine was substituted. Progress of the ulcer was immediately arrested, and the condition of the eye remained stationary. At no time did a hypopyon develop. Two weeks later the area involved was cauterized with the actual cautery, and afterward the ulcer spread so that it involved the pupillary area of the cornea. The eye then healed slowly, and two months after recovery an optic iridectomy was performed. The final vision in this eye was 20/60, with a correcting lens.

CASE 3.—The patient was seen two days after the removal of a foreign body from his eye. An ulcer about 4 mm. in diameter was present directly in the center of the cornea, and a small hypopyon was present. The treatment used in the preceding 2 cases was begun. On the second day sulfapyridine was substituted for sulfathiazole. In spite of all treatment, however, the ulcer progressed slowly and terminated in panophthalmitis. The eyeball was subsequently enucleated.

CASE 4.—Examination the day after removal of a foreign body from the patient's eye revealed a small ulcer in the center of the cornea. The patient received an initial dose of sulfathiazole and thereafter was treated with sulfapyridine in addition to receiving local therapy. After twenty-four hours the ulcer had progressed slightly, so that it involved the whole pupillary area of the cornea. Progress then stopped,

and the ulcer healed, leaving a large, deep corneal scar. At no time was there any involvement of ocular tissues aside from the cornea. Because of the resulting corneal scar, however, useful vision was not retained.

The reason for beginning treatment with sulfathiazole and then abandoning this drug in favor of sulfapyridine should be explained. After the bacteriologic diagnosis was confirmed, Dr. Lepard was asked to see the patients, and it was at his suggestion that the change was made. Several reports have since been published in the ophthalmic literature stating that the concentration of sulfapyridine in the cornea and in the aqueous is about two-thirds or three-fourths that in the blood while the concentration of sulfathiazole is only about one-fifth that in the blood. Therefore, it would seem that if these drugs are to have any beneficial effect at all, sulfapyridine will be much more effective than sulfathiazole. My results might conceivably have been better had sulfapyridine been used from the beginning.

The only conclusion at which one may arrive from a study of these and Dr. Lepard's cases is that sulfapyridine is a valuable adjunct to the usual means of treating corneal ulcer due to B. pyocyaneus.

DR. C. W. LEPARD, Detroit: I wish to thank the discussers for presenting their cases. The rapidity with which infection due to B. pyocyaneus may spread into the eye has been clearly described, and the importance of immediate treatment of this condition has been duly emphasized.

I had the privilege of seeing the 4 patients to whom Dr. Cooper referred, and they presented the typical history and findings of this infection. I am also pleased that Dr. Cooper mentioned the work of Scheie and Souders in which they demonstrated the diffusion of sulfathiazole into the aqueous: The diffusion of sulfathiazole was only 18 per cent, while that of sulfapyridine was 81 per cent. I wish to emphasize again the preference for sulfapyridine. Sulfanilamide also has a high rate of diffusion in the treatment of this infection.

Book Reviews

Obstruction of the Central Retinal Vein. By F. C. Cerboni. Pp. 102, with 17 illustrations. Buenos Aires, 1941.

The author, in his graduation thesis for the University of Buenos Aires, reviews the anatomy, histopathology and history of clinical observation on obstruction of the central retinal vein and reports his findings in 4 cases. The illustrations are chiefly photographs of fundus conditions, and there is a bibliography. Various theories of pathogenesis are reviewed. While arteriosclerosis seems to be the most common factor, the condition is due in a certain number of cases to syphilis and in others to inflammation in or about the vein resulting from foci of infection in the teeth, in the tonsils or elsewhere. Of the author's 4 cases, arteriosclerosis with hypertension was present in 3, while in the other syphilis was considered probable, the patient being 38 years of age, with a positive Kahn reaction. This was the only case in which considerable improvement occurred, vision returning to ⅔ after three months. Secondary glaucoma occurred in 1 of the cases, but surgical intervention was not required, as the eye remained free of pain. The only treatment given in the author's cases was use of acetylcholine, dietary measures and administration of ethylmorphine hydrochloride, with miotics in the case in which glaucoma developed and antisyphilitic treatment in the case of syphilis. In his review of therapy, the recent cases in which treatment was given with heparin are discussed. Complete recovery of vision has occurred in only a few cases, 7.5 per cent according to Wessely. Glaucoma develops in from 13 to 23 per cent of cases and is especially difficult to control. The tension at the onset is usually below normal in the affected eye, the difference between the two eyes varying from 2 to 9 mm. in the author's cases. According to Wessely the prognosis is less favorable in cases in which there is a greater difference in tension between the two eyes. It is less favorable when the patient has a higher blood pressure and more favorable when the patient is young. The second eye is involved in approximately 6 per cent of cases, and Moore's statistics indicate that 41.6 per cent of affected persons die within six years as a result of cerebral vascular accidents.

SANFORD R. GIFFORD.

Two Anatomical Charts of the Eye in Color. Each chart mounted on linen with rollers. Price, $4.75. Toronto, Canada: Rudolf Schick, 1942.

The first chart shows the refractive conditions in the emmetropic, the hypermetropic and the myopic eyeball. Each drawing measures 26 by 12 inches. The second chart shows a view of the eye from in front. To reveal the protective mechanism the skin has been removed so that the deeper structures are visible. The second part is a transverse section of the eyeball with the optic nerves and skull viewed from above, to represent sight as a function of the brain. The drawings measure 26 by 18 inches. The charts illustrate clearly the selected conditions and will be found useful in teaching. There will undoubtedly be room for additional charts, as there are many other views of the eyeball and of its neighboring structures which lend themselves well to chart illustration.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 17 Horseferry Rd., London, England.

Pan-American Congress of Ophthalmology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretaries: Dr. Conrad Berens, 35 E. 70th St., New York. Dr. M. E. Alvaro, 1511 Consolacao, São Paulo, Brazil.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. W. Clark Souter, 9 Albyn Pl., Aberdeen, Scotland.
Secretary: Dr. Frederick Ridley, 12 Wimpole St., London, W. 1.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Hungarian Ophthalmological Society

President: Prof. I. Iwre, Budapest.
Assistant Secretary: Dr. Stephen de Grósz, University Eye Hospital, Mária-utca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Place: Birmingham and Midland Eye Hospital.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. A. MacRae, 6 Jesmond Rd., Newcastle-upon-Tyne, England.
Secretary: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Place: Manchester, Bradford, Leeds, Newcastle-upon-Tyne, Liverpool and Sheffield, in rotation. Time: October to April.

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA

President: Dr. A. James Flynn, 135 Macquarie St., Sydney.
Secretary: Dr. D. Williams, 193 Macquarie St., Sydney.

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the secretary, Dr. Mohammed Khalil.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1, England.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Rd., Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. A. J. Ballantyne, 11 Sandyford Pl., Glasgow, C. 3, Scotland.
Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

SÃO PAULO SOCIETY OF OPHTHALMOLOGY

President: Dr. Jacques Tupinambo, 258 Inglezes, São Paulo, Brazil.
Secretary: Dr. Silvio de Almeido Toledo, Enfermaria Santa Luzia, Santa Casa de Misericordia, Cezario Motta St.—112, São Paulo, Brazil.

SOCIEDAD OFTALMOLOGIA DEL LITORAL, ROSARIO (ARGENTINA)

President: Prof. Dr. Carlos Weskamp, Laprida 1159, Rosario.
Secretary: Dr. Juan M. Vila Ortiz, Córdoba 1433, Rosario.
Place: Rosario. Time: Last Saturday of every month, April to November, inclusive.
All correspondence should be addressed to the President.

SOCIEDADE DE OPHTHALMOLOGIA E OTO RHINO-LARYNGOLOGIA DA BAHIA

President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil.
Secretary: Dr. Adroaldo de Alencar, Brazil.
All correspondence should be addressed to the President.

SOCIETÀ OFTALMOLOGICA ITALIANA

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome.
Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome.

Société Française d'Ophtalmologie

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

Society of Swedish Ophthalmologists

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 Ill tr., Stockholm, Sö.

Tel Aviv Ophthalmological Society

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. Conrad Berens, 35 E. 70th St., New York.
Secretary: Dr. Derrick Vail, 441 Vine St., Cincinnati.
Place: San Francisco.

American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology

President: Dr. Ralph I. Lloyd, 14–8th Ave., Brooklyn.
President-Elect: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

American Ophthalmological Society

President: Dr. Hunter H. McGuire, 105 N. Braddock St., Winchester, Va.
Secretary-Treasurer: Dr. Walter S. Atkinson, 129 Clinton St., Watertown, N. Y.
Place: Hot Springs, Va. Time: June 1943.

Association for Research in Ophthalmology, Inc.

Chairman: Dr. John Evans, 23 Schermerhorn St., Brooklyn.
Secretary-Treasurer: Dr. C. S. O'Brien, University Hospital, Iowa City.

Canadian Medical Association, Section on Ophthalmology

President: Dr. Alexander E. MacDonald, 170 St. George St., Toronto.
Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto.

Canadian Ophthalmological Society

President: Dr. J. Vaillancourt, 46 St. Louis St., Quebec.
Secretary-Treasurer: Dr. Alexander E. MacDonald, 421 Medical Arts Bldg., Toronto.

National Society for the Prevention of Blindness

President: Mr. Mason H. Bigelow, 1790 Broadway, New York.
Secretary: Miss Regina E. Schneider, 1790 Broadway, New York.
Executive Director: Mrs. Eleanor Brown Merrill, 1790 Broadway, New York.

SECTIONAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. Charles W. Barkhorn, 223 Roseville Ave., Newark.
Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. D. G. Hugo, 130 Main St., Oshkosh.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.
Time: October 1942.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. William D. Rowland, 84 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Ralph A. Fenton, 1020 S. W. Taylor St., Portland, Ore.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: Portland, Ore. Time: May 11-14, 1942.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Spencer S. Howe, 103 E. Holly St., Bellingham, Wash.
Secretary-Treasurer: Dr. Barton E. Peden, 419 Stimson Bldg., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. Sheldon Clark, 27 E. Stephenson St., Freeport, Ill.
Secretary-Treasurer: Dr. Harry R. Warner, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. M. Kessler, 311 Center Ave., Bay City, Mich.
Secretary-Treasurer: Dr. J. H. Curts, 330 S. Washington Ave., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. C. Davis, 1615 Howard St., Omaha.
Secretary-Treasurer: Dr. J. E. Dvorak, 408 Davidson Bldg., Sioux Falls, S. D.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. L. Chester McHenry, Medical Arts Bldg., Oklahoma City.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville, S. C.
Place: Richmond, Va. Time: November 1942.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

President: Dr. H. L. Brehmer, 221 W. Central Ave., Albuquerque, N. Mex.
Secretary: Dr. A. E. Cruthirds, 1011 Professional Bldg., Phoenix, Ariz.
Place: Albuquerque, N. Mex. Time: November 1942.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. W. M. Dodge, 716 First National Bank Bldg., Battle Creek.
Secretary-Treasurer: Dr. Kenneth Lowe, 25 W. Michigan Ave., Battle Creek.
Time: Last Thursday of September, October, November, March, April and May.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. H. Bailey, 29 S. Oakland Ave., Sharon.
Secretary-Treasurer: Dr. J. McClure Tyson, Deposit Nat'l Bank Bldg., DuBois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. R. R. Kirkpatrick, 6th and Walnut Sts., Texarkana, Ark.
Secretary-Treasurer: Dr. Raymond C. Cook, 701 Main St., Little Rock.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. William M. Bane, 1612 Tremont Pl., Denver.
Secretary: Dr. Harry Shankel, Republic Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. Edward N. DeWitt, 836 Myrtle Ave., Bridgeport.
Secretary-Treasurer: Dr. Henry L. Birge, 179 Allyn St., Hartford.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. E. N. Maner, 247 Bull St., Savannah.
Secretary-Treasurer: Dr. C. K. McLaughlin, 567 Walnut St., Macon.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. F. McK. Ruby, Union City.
Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.
Place: French Lick. Time: First Wednesday in April.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. K. Von Lackum, 117–3d St. S. E., Cedar Rapids.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. Henry N. Blum, 912 American Bank Bldg., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Robert H. Fraser, 25 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. G. Laird, 114 Fulton St., Grand Rapids.

Minnesota Academy of Ophthalmoloy and Otolaryngology

President: Dr. Erling Wilhelm Hansen, 78 S. 9th St., Minneapolis.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. William J. Marshall, 335 Owen St., Missoula.
Secretary: Dr. Fritz D. Hurd, 309 Medical Arts Bldg., Great Falls.

Nebraska Academy of Ophthalmology and Otolaryngology

President: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. John Peterson, 1307 N St., Lincoln.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Edgar P. Cardwell, 47 Central Ave., Newark.
Secretary: Dr. Arthur E. Sherman, 243 S. Harrison St., East Orange.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Searle B. Marlow, 109 S. Warren St., Syracuse.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. M. R. Gibson, Professional Bldg., Raleigh.
Secretary: Dr. Vanderbilt F. Couch, 105 W. 4th St., Winston-Salem.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. G. A. Larson, 114 Roberts St., Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 516–6th St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.
Secretary-Treasurer: Dr. R. S. Fixott, 1020 S. W. Taylor St., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. L. Sanders, 222 N. Main St., Greenville.
Secretary: Dr. J. H. Stokes, 125 W. Cheves St., Florence.
Place: Columbia. Time: November 1942.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Wesley Wilkerson, 700 Church St., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. E. L. Goar, 1300 Walker Ave., Houston.
Secretary: Dr. Dan Brannin, 929 Medical Arts Bldg., Dallas.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. Everett B. Muir, Boston Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Earl H. Phillips, 623 Judge Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Mortimer H. Williams, 30½ Franklin Rd. S. W., Roanoke.
Secretary-Treasurer: Dr. Meade Edmunds, 34 Franklin St., Petersburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George E. Ferguson, Second National Bldg., Akron, Ohio.
Secretary-Treasurer: Dr. D. F. Mathias, First Central Tower, Akron, Ohio.
Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Stacy C. Howell, 144 Ponce de Leon Ave. N. E., Atlanta, Ga.
Secretary: Dr. Lester A. Brown, 478 Peachtree St. N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., second Wednesday of each month, from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Ernst Bodenheimer, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Thomas R. O'Rourk, 104 W. Madison St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to March.

BIRMINGHAM EYE, EAR, NOSE AND THROAT CLUB

President: Each member, in alphabetical order.
Secretary: Dr. Luther E. Wilson, 919 Woodward Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. Maurice Wieselthier, 1322 Union St., Brooklyn.
Secretary-Treasurer: Dr. Harold F. Schilback, 142 Joralemon St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

CHATTANOOGA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Each member, in alphabetical order.
Secretary: Dr. Douglas Chamberlain, Chattanooga Bank Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. Sanford Gifford, 720 N. Michigan Ave., Chicago.
Secretary-Treasurer: Dr. Vernon M. Leech, 55 E. Washington St., Chicago.
Place: Chicago Towers Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

CINCINNATI GENERAL HOSPITAL OPHTHALMOLOGY STAFF

Chairman: Dr. D. T. Vail, 441 Vine St., Cincinnati.
Secretary: Dr. A. A. Levin, 441 Vine St., Cincinnati.
Place: Cincinnati General Hospital. Time: 7:45 p. m., third Friday of each month except June, July and August.

CLEVELAND OPHTHALMOLOGICAL CLUB

Chairman: Dr. W. J. Abbott, 10515 Carnegie Ave., Cleveland.
Secretary: Dr. L. V. Johnson, 2065 Adelbert Rd., Cleveland.
Time: Second Tuesday in October, December, February and April.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Francis H. Adler, 313 S. 17th St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman: Dr. C. D. Postle, 240 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Hugh C. Thompson, 289 E. State St., Columbus, Ohio.
Place: The Neil House. Time: 6 p. m., first Monday of each month.

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. F. K. Stroud, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. Arthur Padilla, 414 Medical Professional Bldg., Corpus Christi, Texas.
Time: Second Friday of each month from October to May.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. A. Nelson, 4105 Live Oak St., Dallas, Texas.
Secretary: Dr. S. F. Harrington, 921 Medical Arts Bldg., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. H. C. Schmitz, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Byron M. Merkel, 604 Locust St., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Harvey E. Dowling, 2414 Eaton Tower, Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Detroit Ophthalmological Society

President: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Leland F. Carter, 1553 Woodward Ave., Detroit.
Place: Club rooms of Wayne County Medical Society. Time: Third Thursday of each month from November to April, inclusive.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. James M. Dunn, 1352 Union St., Schenectady.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Eastern Pennsylvania Association of Eye, Ear, Nose and Throat Physicians

President: Dr. James E. Landis, 232 N. 6th St., Reading.
Secretary-Treasurer: Dr. Sterling F. Mengel, 612 W. Market St., Pottsville.
Time: Last week in April each year.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. W. R. Thompson, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. A. E. Jackson, 602 W. 10th St., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Ophthalmological and Oto-Laryngological Section

President: Dr. Felician J. Slataper, 1110-1111 Medical Arts Bldg., Houston, Texas.
Secretary: Dr. Theo. L. Holland, 611 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. John I. Garret, 57 Stokes Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Homer Beal, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. Desmond Curran, Federal Reserve Bank Bldg., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Harold Snow, 614 S. Pacific Ave., San Pedro, Calif.
Secretary-Treasurer: Dr. Oliver R. Nees, 508 Times Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. John Osburn, 523 W. 6th St., Los Angeles.
Secretary-Treasurer: Dr. Colby Hall, 1136 W. 6th St., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye and Ear Society

President: Dr. Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. W. Fish, 321 W. Broadway, Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Lower Anthracite Eye, Ear, Nose and Throat Society

Chairman: Each member in alphabetical order.
Secretary: Dr. James J. Monohan, 31 S. Jardin St., Shenendoah, Pa.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. E. J. Cummings, 1835 I St. N. W., Washington.
Secretary: Dr. P. S. Constantinople, 1835 I St. N. W., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month from September to May.

Milwaukee Oto-Ophthalmic Society

President: Dr. John W. Truitt, 161 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. Ralph T. Rank, 238 W. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Montgomery County Medical Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. J. Rosenbaum, 1396 Ste. Catherine St. W., Montreal, Canada.
Secretary: Dr. L. Tessier, 1230 St. Joseph Blvd. E., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Secretary: Dr. Guy Maness, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Haven Ophthalmological Society

President: Dr. William H. Ryder, 185 Church St., New Haven, Conn.
Secretary: Dr. Frederick A. Wies, 255 Bradley St., New Haven, Conn.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Secretary: Dr. Mercer G. Lynch, 1018 Maison Blanche Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to May.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Kaufman Schlivek, 1016–5th Ave., New York.
Secretary: Dr. Brittain Payne, 896 Madison Ave., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President: Dr. Isidore Givner, 108 E. 66th St., New York.
Secretary: Dr. Benjamin Esterman, 983 Park Ave., New York.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. E. Leatherock, Cushing, Okla.
Secretary: Dr. Harry C. Ford, 1014 Medical Arts Bldg., Oklahoma City.
Place: University Hospital. Time: Second Tuesday of each month from September to May.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Leo P. Coakley, 918 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. L. Markowitz, 16 Church St., Paterson, N. J.
Secretary-Treasurer: Dr. A. John Reinhorn, 302 Broadway, Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Secretary: Dr. Wilfred E. Fry, 1819 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. J. Clyde Markel, 200–9th St., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

READING EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Solon L. Rhode, 238 N. 6th St., Reading, Pa.
Secretary: Dr. Paul C. Craig, 232 N. 5th St., Reading, Pa.
Place: Wyomissing Club. Time: 6:30 p. m., third Wednesday of each month from October to July.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. F. Bryce, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. E. J. Avery, 11 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.

St. Louis Ophthalmic Society

President: Dr. Leslie C. Drews, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. H. Rommel Hildreth, 823 Metropolitan Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmo-Oto-Laryngological Society

President: Dr. Dan Russell, 705 E. Houston St., San Antonio, Texas.
Secretary-Treasurer: Dr. P. G. Bowen, 315 Camden St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Fred Boyle, 490 Post St., San Francisco.
Secretary: Dr. Frank Hand, 450 Sutter St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.
Secretary-Treasurer: Dr. Kenneth Jones, Medical Arts Bldg., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Louis A. Parsell, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Robert L. Pohl, 407 Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Roy Seeley Moore, 1704 State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. H. Blaisdell, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toledo Eye, Ear, Nose and Throat Society

Chairman: Dr. John D. Skow, 2001 Collingwood Blvd., Toledo, Ohio.
Secretary: Dr. S. H. Patterson, 125–15th Ave., Toledo, Ohio.
Place: Toledo Club. Time: Each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. L. Conner Moss, 3260 Wilson Blvd., Arlington, Va.
Secretary-Treasurer: Dr. Harold R. Downey, 1740 M St. N. W., Washington, D. C.
Place: Medical Society of District of Columbia Bldg., 1718 M St N. W. Time: 7:30 p. m., first Monday in November, January, March and May.

Wilkes-Barre Ophthalmological Society

Chairman: Each member in turn.
Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.
Place: Office of chairman. Time: Last Tuesday of each month from October to May.